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GUANETHIDINE—REPORT OF A CLINICAL TRIAL*

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GUANETHIDINE‡ is a synthetic antihypertensive agent whose mode of action is thought to be by interference with the release of noradrenaline from the sympathetic nerve endings. The pharmacological effect in therapeutic doses in man is closely similar to that of bretylium tosylate. Both compounds show sympathicolytic effect without the symptoms and signs of ganglionic blockade. Blood pressure is reduced, orthostatic hypotension occurs, bradycardia may be striking and bowel frequency is common. The uncomfortable to potentially dangerous constipation and urinary slowing, characteristic of ganglionic blockade, does not occur in doses within the therapeutic range.[1, 8]

In contrast to the hemodynamic effects of the ganglionic blocking agents, consisting of a reduction of cardiac output, Taylor and Donald[7] have shown that bretylium, and to a lesser extent guanethidine, would appear to owe their blood pressure lowering effect to a loss of effective vasoconstriction in non-exercising muscle. With bretylium they found a consistent rise in resting cardiac output and an increase in pulmonary vascular resistance after intravenous administration. This effect was not observed with guanethidine. In their study they found the fall in blood pressure after exercise, in the patient taking bretylium tosylate, to be marked in degree, while they did not observe a similar effect after guanethidine. This latter effect has, however, been observed by Dollery *et al.*[8] Pigeon *et al.*,[4] and by the author of this report, although not in all patients. Dollery *et al.*[8] have observed maximum hypotensive effect to occur in the morning, regardless of the time of day that guanethidine was given. In 20 patients of the author this phenomenon was observed only once to a significant degree. Dollery,[8] in several of his 80 patients, found it advisable to give a dose of pempidine* at bedtime, in order to achieve more uniform control of blood pressure, since he reports in several cases that when the morning weakness has been disregarded, the patients develop effort syncope later in the day. While the author has used pempidine as an adjunct in the induction of hypotension, it was not found advisable or necessary to use such drugs concurrently on a long-term basis.

Guanethidine is available in tablets of 10 to 25 mg. strength. Patient acceptance of this medication is good. The usual therapeutic dose requires relatively few tablets per day and the taste of the broken tablet is not unpleasant.

The onset of action is slow and is measured in days rather than fractions of an hour. The hypotensive effect is prolonged, indicating duration of overdose symptoms up to a week. Rather wide variation in the range of effective therapeutic dose between patients seems common, but the maintenance dose for any one patient seems relatively constant. The author begins therapy with initial small doses, 10 to 25 mg. per day, increasing by increments of 10 to 25 mg. per day every third day until the therapeutic effect is achieved. With this routine the therapeutic dose has been the same as the maintenance dose. The development of resistance, necessitating a significant increase in the amount of drug given, has not been observed in our patients. Some 30% of this group have required slight adjustment of dosage, usually upward, after leaving hospital and resuming activity (Fig. 1).

In the presence of an urgent indication for rapid reduction of severe diastolic hypertension, as in hypertensive encephalopathy or impending stroke, the author has initiated therapy with guanethidine and the ganglionic blocking agent, pempidine, used simultaneously. The initial dose of guanethidine is 25 mg. per day, as it is for less severely hypertensive patients. The dose is increased in a step-wise fashion, as described above. The dose of pempidine is titrated as with any relatively short-acting ganglionic blocking agent. The initial dose of 10 mg. by mouth is repeated in 2 hours if no

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*Pempidine is not commercially available in Canada. Supplies were generously provided by Ayerst, McKenna & Harrison Limited and the Imperial Chemical Company through Drs. Leighton Smith and A. S. R. Stewart respectively.

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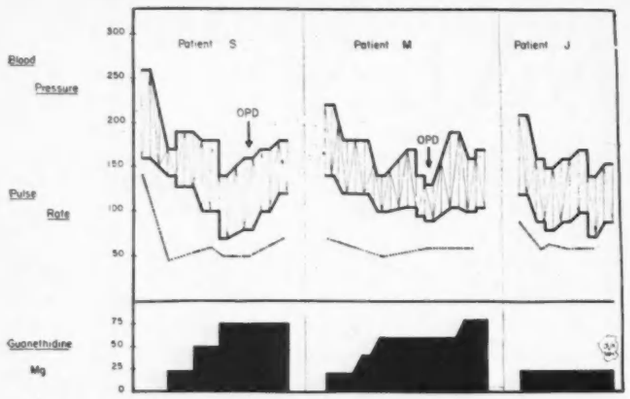

Fig. 1.—Three female patients, two of whom required a slight increase in guanethidine dosage when resuming activities.

hypotensive effect is apparent. This is continued until a therapeutic dose level is reached. Pempidine is then given every 6 to 8 hours as indicated by a significant rise in diastolic blood pressure above the therapeutic floor established, short of toxic symptoms. Before each increase in guanethidine dosage, the intervals between pempidine administration are increased, while the behaviour of the diastolic blood pressure is being observed. If no significant rise occurs 8 to 10 hours after the previous dose of pempidine, it is safe to infer that guanethidine is exerting a pharmacological effect, and pempidine therapy is discontinued. This drug combination shows no synergism or enhanced toxicity (see Table II, Case E.F.; also Figs. 2 and 3).

Pempidine is a totally absorbed ganglionic blocking agent[6] which produces dry mouth, blurred vision, constipation, urinary slowing and impotence, as do all other members of this group. Its therapeutic effect is more rapid in onset and shorter in duration than that of mecamylamine. With pempidine the author has not observed the tremor

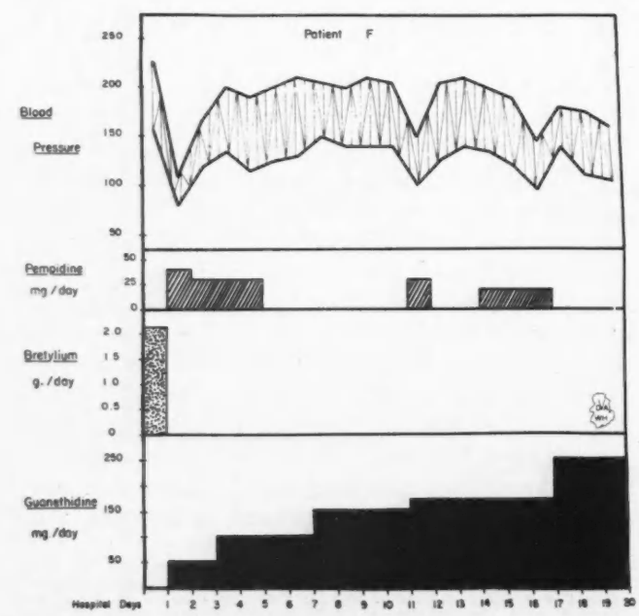

Fig. 2.—Initial resistance to bretylium, with the use of pempidine in conjunction with guanethidine titration to control excesses of hypertension.

and psychic disturbances which sometimes accompany the use of mecamylamine. Nevertheless toxic effects may be formidable. In one young patient (C.H.) an ileus developed during administration of this combination of guanethidine and pempidine, and the patient required laparotomy for suspected bowel perforation.

Despite the basic similarity in clinical effects of bretylium and guanethidine, certain significant differences are observed in the subjective and objective effects resulting from its administration (Table I).

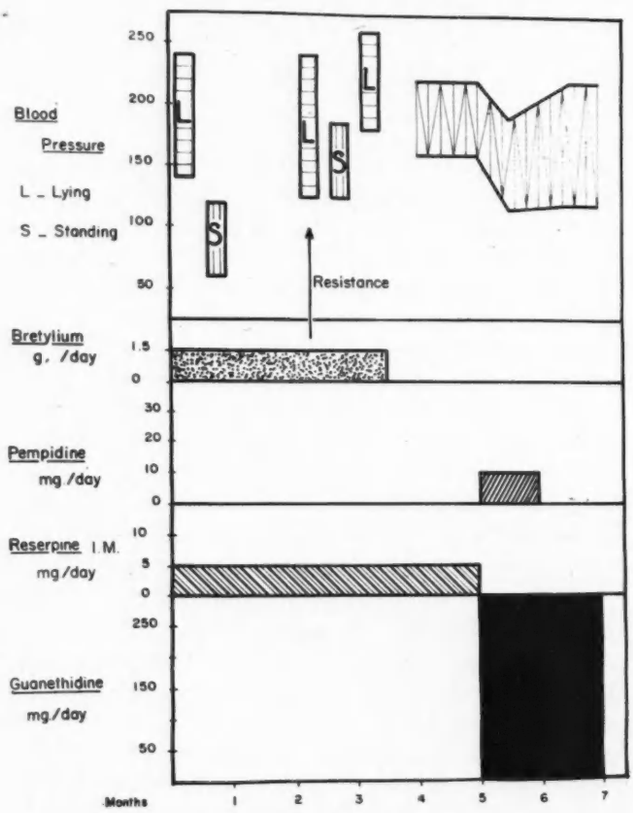

Fig. 3.—Again, resistance to bretylium, with combined control by pempidine and guanethidine.

In the Cardiac Service at Westminster Hospital and in the author's consulting practice, 20 patients have been studied before and during treatment with guanethidine. The shortest period of observation was three weeks, the longest nine months. In three patients sufficient urgency of treatment existed to justify the induction of antihypertensive

TABLE I.—CONTRAST BETWEEN BRETYLIUM AND GUANETHIDINE

	Bretylium	*Guanethidine*
Onset of effect	Less than 1 hour	3–5 days
Duration of effect	6–8 hours	3–7 days
Bradycardia	+	+ to + + +
Diarrhea	0 to +	+ to + + + +
Indigestion	Severe in 4 of 10	1 in 15 3 in 28 (Pigeon *et al.*)
Parotid pain	0 to intolerable	Not observed
Weakness	In 5 of 10	Mild in 3 of 15
Severe orthostasis	In 4 of 10	Not observed
Impotence	Not observed	Not observed

TABLE II.—SUMMARY OF CASES

Name	*Age*	*B.P. pre-treatment*	*B.P. treated*	*Blood urea (mg. %)*	*ECG pattern*	*Guanethidine dose/day*	*Previous drug treatment*	*Complications*	*Diagnosis*	*Treatment concurrent with guanethidine*
C.H.	25	200/140	220/140 (L) 170/115 (S)	130	Left ventricular hypertrophy	175 mg.	I.M. reserpine	G.I. hemorrhage	Polyarteritis nodosa	None
A.W.	64	210/130	155/90 (L) 140/80 (S)	51	Left ventricular strain	60 mg.	Ganglionic blockers since 1954	Prostatic hypertrophy		Digitalis
E.F.	42	225/160	180/120 (L) 180/110 (S)	39	Left axis deviation	200 mg.	Bretylium (resistant) 2.4 g./day	Hypertension diarrhea	Encephalopathy	Induced with pempidine
R.M.	48	240/132	224/110 (L) 148/90 (S)	45	Normal	50 mg.	None	Diarrhea	Transient hemiplegia	Codeine
F.C.	47	240/130	185/105 (L) 140/70 (S)	51	Left ventricular hypertrophy	100 mg.	None	Diarrhea	Emphysema, auricular fibrillation	Quinidine maintenance
C.G.	38	190/140	180/115 (L) 160/110 (S)	30	Left axis deviation	50 mg.	None	None	Angina	None
R.J.	65	260/145	140/90 (L) 120/75 (S)	36	Left ventricular strain	40 mg.	Digitalis. Thiomerin	None	Unilateral renal atrophy	Digitalis and thiomerin continued
F.A.C.	66	250/135	190/105 (L)	57	Intermittent left B.B.B.	40 mg.	None	None	Old stroke. Renal calculi	None
E.K.	61	190/150	180/100 (L) 135/80 (S)	42	Left ventricular strain	60 mg.	Thiomerin. Digitalis	None		None—failure-free
G.M.	65	260/140	205/130 (L)	50–80	Auricular fibrillation	10-150 mg.	Ganglionic blockers	Deceased	Malignant hypertension	Induced with pempidine, slow response to guanethidine
G.W.	67	240/130	200/105 (L) 160/90 (S)	36	Left ventricular strain	75 mg.	None	None		None
F.H.	63	200/130	160/110 (L)	45	Arborization block	60 mg.	None	Falls from postural syncope	Recent stroke	None
J.E.H.	60	210/120	165/110 (L) 145/100 (S)	27	Left ventricular strain	70 mg.	None	None	Simple schizophrenia	None
J.McD.	70	200/115	150/90 (L) 125/75 (S)	39	Left ventricular hypertrophy	35 mg.	Rauwolfia	None	In failure	None Failure clearing
Mrs. S.	64	240/160	170/105 (L) 210/100 (S)	40	Left axis deviation	75 mg.	None	Severe diarrhea	Severe epistaxis	Atropine
Mrs. Mc.	42	230/140	160/100 (L) 160/95 (S)	30	Normal	80 mg.	Reserpine. Thiozide	Moderate diarrhea	Encephalopathy	None
Mrs. J.	52	210/120	135/75 (L)	50	Normal	25 mg.	Hydralazine	None	Stroke 5 years ago	None
A.D.	71	250/130	220/100 (L) 160/90 (S)	35	Left ventricular strain	75 mg.	Mecamylamine	Ankle swelling		None
H.Mc.	53	190/120	180/100 (L) 160/90 (S)	30	Left ventricular hypertrophy	30 mg.	Mecamylamine. Hydralazine	None	In failure	Induced with pempidine
R.S.	54	170/110	200/110 (L) 120/80 (S)	51	Non-specific T-wave inversion	50 mg.	Bretylium	None	Diabetes. In failure	Digitalis

therapy with the combination of pempidine and guanethidine. Table II summarizes the clinical characteristics of these patients and results of treatment.

The following case reports are selected to show the relative safety of this method of treatment, the disadvantage in the tardiness of onset of effect of guanethidine and the contrast between bretylium and guanethidine.

CASE 1, C.H. This 24-year-old man had an established diagnosis of polyarteritis nodosa, having had three abdominal perforations with surgical closure. He had been treated with various ganglionic blocking agents and for a prolonged period with intramuscular reserpine, 2.5 mg. twice daily. In December 1959, he was given a trial with bretylium tosylate which brought about fairly marked orthostatic hypotension but little alteration in his recumbent blood pressure. He became agitated and discontinued bretylium on his own and returned to intramuscular reserpine.

He was admitted in July 1960 with severe gastrointestinal bleeding, the site of which was not determined. He had not been receiving corticosteroids. He was given guanethidine, 300 mg. per day, with blood pressure control as indicated; there were no toxic manifestations. In an effort to reduce his blood pressure he was given a combination of pempidine and guanethidine. Within a few hours of the first dose of pempidine he developed abdominal pain, distension, vomiting and faint peristalsis with pinched facial appearance. The abdomen was explored surgically within eight hours and no evidence of bowel perforation was detected. His convalescence was satisfactory, and he has continued antihypertensive treatment with guanethidine without toxic manifestations.

CASE 2, F.C. This 47-year-old man was admitted to the Heart Service of Westminster Hospital on June 9, 1960, for investigation of hypertension. He had no symptoms at the time of admission, but some five years previously he had had severe occipital headaches,

which occurred in the morning and cleared during the day. At the same time he noticed a pulsatile mass at the root of his neck on the right side. He had experienced slight shortness of breath on exertion for the past year. He was treated elsewhere for a short time, evidently with antihypertensive therapy, the nature of which is not known.

He was a short, broad, fairly muscular, sallow man, looking older than his stated age, with an average blood pressure of 240/130. His eyegrounds showed arteriovenous nicking, but no hemorrhage or exudate. The apex impulse was in the anterior axillary line. The heart action was vigorous, with a systolic murmur at the apex and over the aortic area. The lung bases were clear. The liver was not enlarged. The neck mass was found to be a tortuosity of the common carotid artery. Phenolsulfonphthalein excretion was 41% in the first hour. Blood urea was 51 mg. %. He was given guanethidine beginning June 22, 1960, and after four days he responded minimally to 25 mg. a day, to which was added chlorothiazide, without any enhancement of effect. Therefore the dose of guanethidine was increased step-wise to 100 mg. daily. At about this time the patient developed auricular fibrillation, which reverted to normal sinus rhythm after administration of 12 grains of quinidine per day. There was no evidence of myocardial necrosis, and he was sent home. He is now working as a machinist and when last seen on September 1, 1960, his recumbent blood pressure was 184/104 mm. Hg and his standing blood pressure was 142/70. He passed 3 to 5 stools daily and was given atropine, without a change being made in the daily dose of 100 mg. of guanethidine.

CASE 3, E.K. This 61-year-old patient was admitted to the Cardiac Service of Westminster Hospital on May 12, 1960, for investigation and treatment of hypertension. He had recently had a respiratory infection with recurrence of dyspnea, wheezing and nocturnal dyspnea after this attack, accompanied by a gain in weight. He was a stout, emphysematous man with an average blood pressure of 190/150. The heart was slightly enlarged to the left with a presystolic, apical gallop, but no murmur. The urine showed a specific gravity of 1.021 with rare granular casts and a blood urea value of 42 mg. %. He was non-diabetic.

In view of the possibility of complicating left ventricular failure, he was placed at once on antihypertensive therapy with guanethidine; a total dose was reached of 60 mg. per day in divided doses (35 mg. in the morning and 25 mg. in the evening). On May 31, 1960, he was sent home on this therapy and was seen as an outpatient on July 30, 1960, with a recumbent blood pressure of 190/110 and a standing blood pressure of 110/70. The dose was reduced to 40 mg. and on August 3, 1960, the recumbent blood pressure was 180/100 and the standing pressure 135/80. He continued with the same dosage and on August 1, 1960, his standing blood pressure had reached 190/110 and recumbent blood pressure 210/120. The dose was increased again to 60 mg. per day.

DISCUSSION

Guanethidine is another recently synthesized compound that has a sympathicolytic effect. Its action is reported to be enhanced by combination with a thiazide.[8]

For therapy the author begins with a small dose, i.e. 10 or 25 mg. per day, increasing it every third day until a satisfactory hypotensive effect is observed, or as in one case, until severe diarrhea supervenes. Some investigators begin therapy with a single dose of 200 to 250 mg. with step-wise, rapid reduction as therapeutic effect is achieved. This method was not favoured. Toxicity would have been induced in the majority of the author's patients, since the largest required dose to date is 300 mg. per day.

Bradycardia was frequently observed, with rates often as low as 45 per minute, but no abnormality of rhythm or ECG deterioration was noted. No evidence of renal, hepatic or hematopoietic toxicity was observed, nor was skin rash or other signs of hypersensitivity. One patient in this series, without previous ulcer symptoms, has recently developed the hunger-pain-food-relief cycle described by Pigeon *et al.*[4] This was anticipated with bretylium,[5] although typical ulcer periodicity and punctuality have not yet appeared in our patients. Urgent and frequent bowel movements are a nuisance, controllable by codeine or belladonna. They seem to be due to a true hypermotility rather than malabsorption, although this was not studied in detail. One instance of transient leg edema was observed, but no clinical heart failure was encountered.

The extremes of orthostatic hypotension, as seen with bretylium in 4 of 10 cases,[5] have not been observed with guanethidine. Weakness of the extremities has occurred and has been a serious complaint in three cases. One woman reported terrifying nightmares; two have reported mild sedative effects. Salivary gland pain has not been encountered. The gradual lowering of blood pressure by guanethidine may well prove an added safety factor in those with coronary or cerebral vascular disease.

SUMMARY

Observations are reported on a series of 20 hypertensive patients treated by guanethidine, either alone or, in cases of malignant hypertension, in concert with pempidine. The effects of guanethidine are produced gradually and dissipate slowly. Severe postural hypotension has not been a problem. Diarrhea and weakness of the limbs have been the most frequent complaints. Peptic indigestion developed in one patient. No evidence of tolerance has yet been observed.

The introduction of selective sympathicolytic drugs is a significant advance in the treatment of severe hypertension and may replace the long-term use of ganglionic blocking drugs. The complications incident to vagus overactivity are already appearing. A combination of the rapid-acting, totally absorbed pempidine with guanethidine in the induction of treatment of malignant hypertension seems safe and of value.

ACKNOWLEDGMENT

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GUANETHIDINE AS THE SOLE AGENT IN THE TREATMENT OF HYPERTENSION*

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BRETYLIUM TOSYLATE[1-5] and guanethidine[6] are two new drugs available for the treatment of hypertension. Both seem to act by inhibiting sympathetic nerve function without provoking the severe side effects of parasympathetic inhibition that are produced by the ganglionic blocking drugs.

In this report the response of 12 hypertensive patients (two with malignant hypertension) to guanethidine is described in detail to demonstrate the effectiveness of this drug as the sole therapeutic agent in severe hypertension and to illustrate the marked differences in individual sensitivity to guanethidine.

CASE REPORTS

CASE 1.—I.S., a 36-year-old man, had been paraplegic since sustaining a compression fracture of the tenth thoracic vertebra in 1943. Automatic bladder function was present and chronic pyelonephritis and renal calculi eventually developed. Severe hypertension had been present since 1953, with normal blood pressure before that year. On admission to hospital in October 1959, his blood pressure was 260/140 mm. Hg, and averaged 230/135 for two weeks without therapy. The optic fundi showed papilledema, hemorrhages, exudate and moderate arteriolar changes. Early heart failure was present. Administration of guanethidine was started on November 9, 1959 (Fig. 1). The blood pressure did not decrease after usual doses of guanethidine and eventually the dose was increased by 100 mg. increments at four-day intervals to a total daily dose of 600 mg., following which a satisfactory decrease in blood pressure was observed. Despite the large dose of guanethidine there were no side effects attributable to the drug. After ten weeks' therapy at this dose level, the blood pressure was in the range of 120-150/70-90 lying, and 130/85 sitting. The patient could not stand, so that no postural symptoms were experienced. Tilting the patient to 60° on a table for ten minutes did not produce any symptoms.

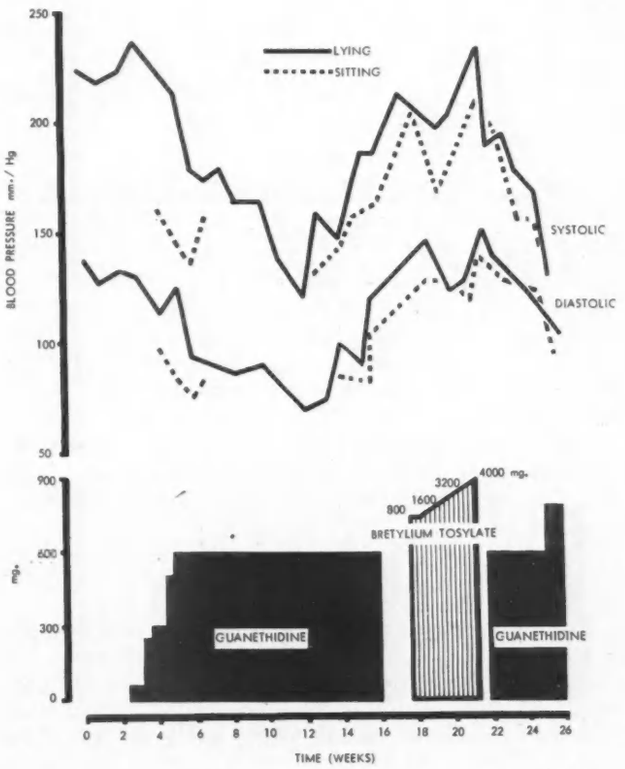

Fig. 1.—Case 1. Blood pressure reduction on large doses of guanethidine and refractoriness to bretylium are indicated.

Guanethidine was discontinued on February 9, 1960. By February 20, the blood pressure was 220/150; bretylium tosylate was then started. The initial dose of this agent was 0.8 g. daily, and at four-day intervals 0.8 g. increments were added to reach a total daily dose of 4.0 g. Nausea and vomiting occurred but lessened despite continued administration of the drug. The blood pressure continued to rise and reached a peak of 263/170 mm. Hg by March 21, 1960. Encephalopathy occurred at this time, manifested by depression, semicoma, generalized twitching and inability to swallow. The bretylium tosylate was discontinued. The patient was given 100 mg. of guanethidine intravenously and in six hours his blood pressure decreased by about 50 mm. systolic and 20 mm. diastolic (Fig. 2). An additional 300 mg. of guanethidine was administered intravenously after this initial blood pressure fall, but the blood pressure did not decrease any further in the next 24 hours. However, the symptoms and signs of encephalopathy cleared within 12 hours after intravenous injection of guanethidine. Oral guanethidine at a dose of 600 mg./day was restarted and increased to 800 mg. a day until the desired response was again

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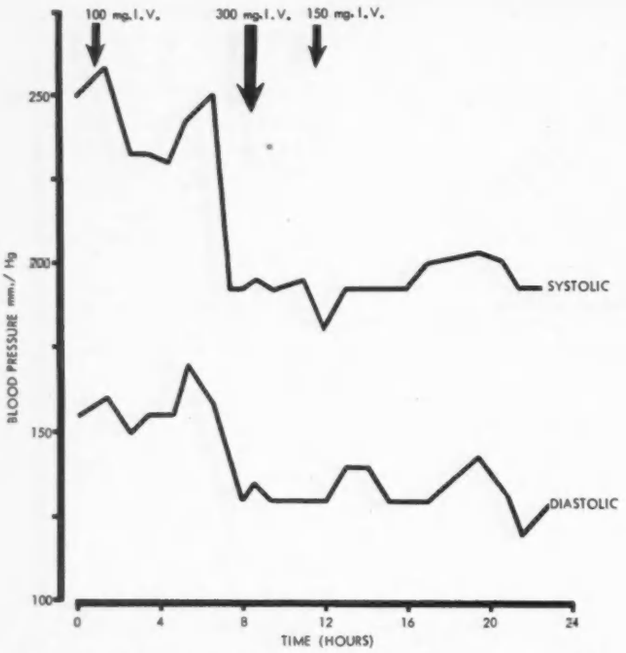

Fig. 2.—Case 1. Response to intravenous guanethidine during hypertensive encephalopathy.

obtained, the blood pressure gradually falling to 150/90 lying and 138/90 sitting. Later it was found that the hypotensive effect could be maintained by half of this dose of guanethidine, if chlorothiazide was added to the therapeutic regimen. The fact that this patient had an indwelling catheter excluded the possibility that variable bladder distension may have affected his blood pressure levels.

Additional Studies

Skin temperature and skin resistance studies were carried out (Table I). While therapy was not given, the finger temperature rose and fell through a normal range with body heating and cooling, suggesting normal sympathetic vasomotor control. While 4.0 g. of bretylium tosylate daily was given, approximately the same range of temperature change of the fingers was obtained with body warming and cooling, suggesting that sympathetic function was still intact. However, while he received guanethidine 800 mg. daily, the control temperatures were much lower and the response to heating and cooling was considerably reduced, suggesting that sympathetic control was partially blocked. Also, the finger temperatures while the patient was taking guanethidine resembled those of the toes, which were not subject to sympathetic control because of this patient's paraplegia. Room temperature during these studies was constant at 20° C. Toe temperature suggested complete absence of sympathetic control prior to any therapy, owing to the paraplegia, and skin resistance studies indicated the absence of sweating from the hips down.

Several attempts at clearance studies were unsatisfactory because of considerable bladder reflux up the dilated ureters. The patient's initial blood urea nitrogen value of 41 mg. % increased to 61 mg. % with the initial reduction of blood pressure, but at final discharge it was 32 mg. % after the blood pressure had been nearly normal for several weeks.

Results of liver function tests, including bromsulfthalein retention, flocculation studies, serum bilirubin, plasma proteins, serum glutamic oxaloacetic transaminase and serum glutamic pyruvic transaminase, were normal after ten weeks of guanethidine therapy at a dose of 600 mg. daily.

Urine guanethidine excretion, estimated by a colorimetric method,[7] was found to be high, indicating that the orally administered drug was being absorbed. The absence of significant blood pressure decrease after a large intravenous dose of guanethidine is further evidence that the large oral dose requirement was attributable to the patient's lack of sensitivity to guanethidine and not to inadequate absorption of the drug.

COMMENT

There were several unusual features in this case. The dose of guanethidine required to produce a satisfactory hypotensive effect was higher than any previously reported. Most patients respond to daily doses of 25 to 100 mg.[6] Whatever the mechanism of the relative insensitivity to this drug, this case afforded excellent opportunity for assessment of guanethidine tolerance and toxicity. No side effects were experienced from the large doses employed. Bladder and bowel function were unchanged, no parasympathetic blocking action was observed, the heart rate remained relatively constant, and no gastric upset was experienced. Hematological and hepatic function studies showed no changes attributable to guanethidine, and the patient's renal function apparently improved.

TABLE I.—SKIN TEMPERATURE STUDY. TEMPERATURES IN °C.

Therapy	*Finger*		*Toes*		*Oral*	
	Control	*Heating*	*Control*	*Heating*	*Control*	*Heating*
None	30.8	33.3	29.3	26	97.6	98.8
Bretylium tosylate	29.2	32.1	26.2	23.5	97.6	99.0
Guanethidine	24.5	26.5	24.5	22.5	97.4	99.4

The failure of bretylium tosylate to reduce blood pressure in some cases has been previously noted.[2, 3] The skin temperature studies indicated that sympathetic function was essentially unaltered by the unusually high doses of bretylium tosylate. Any further increment in the dose of this agent was precluded when hypertensive encephalopathy developed.

Guanethidine is not a suitable agent for use when a rapid reduction in blood pressure is desired. Administration of 100 mg. of guanethidine intravenously produced a sufficient decrease in blood pressure to relieve the encephalopathy but required six hours to do so.

CASE 2.—J.G., a 34-year-old male labourer, suffered from severe hypertension due to chronic pyelonephritis. When first seen he had a blood pressure of 250/140 mm. Hg, early grade 4 retinopathy and left ventricular failure. This degree of hypertension persisted despite treatment with reserpine and chlorothiazide, and these agents were discontinued. Guanethidine therapy was then started with a daily dose of 75 mg. Five days later his blood pressure was 155/100 lying and 110/80 standing, and on one occasion he fainted without warning. Guanethidine was discontinued and four days later his blood pressure rose to 220/120, both lying and standing. With 25 mg. of guanethidine daily, his blood pressure was well controlled (140/90 lying), but he had several fainting spells which occurred without warning. Diarrhea and cramps were experienced. On 12.5 mg. of guanethidine daily, his blood pressure was 188/115 lying and standing; the diarrhea persisted but further episodes of syncope did not occur.

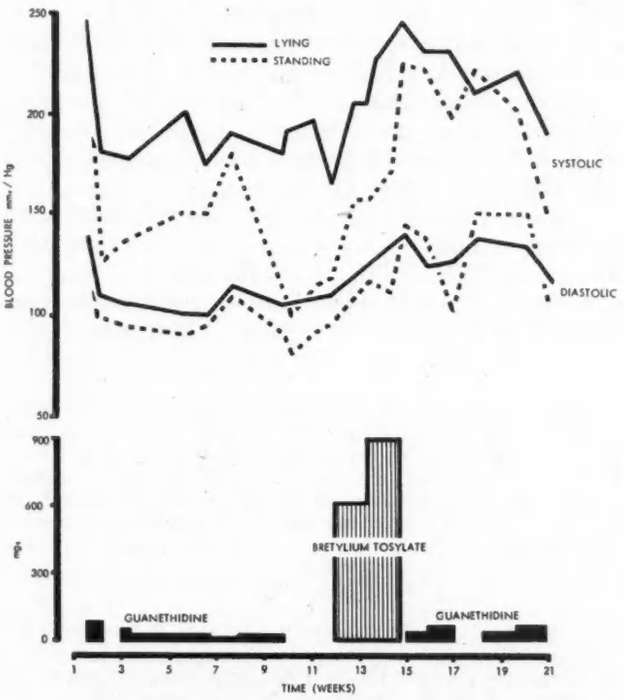

Fig. 3.—Case 2. Blood pressure reduction with small doses of guanethidine, with marked postural effect.

Diarrhea ceased on discontinuation of guanethidine, but the blood pressure then rose to 250/170. The patient was then started on 900 mg. of bretylium tosylate daily. After three weeks on this medication his blood pressure had decreased only slightly (to 246/146) and when generalized weakness, shakiness and vomiting occurred, it was necessary to discontinue the bretylium tosylate. The patient was restarted on 25 mg. of guanethidine daily, with gradual reduction of his blood pressure (see Fig. 3).

The creatinine clearance level decreased from 41 c.c./min. to 21 c.c./min., and the blood urea nitrogen level rose from 37 to 51 mg. % following the reduction of blood pressure with guanethidine. Signs of heart failure disappeared and the retinopathy eventually decreased to grade 2.

This patient was also tested with intravenous guanethidine. A total dose of 75 mg. was given intravenously over a 20-minute period. In one hour the blood pressure (measured by an intra-arterial needle and strain gauge system) rose from 235/112 to 260/140 mm. Hg. The cardiac output (dye dilution curves) increased from 4.3 to 4.6 litres per minute. This increase in blood pressure and cardiac output after parenteral injection of guanethidine has been previously noted in dogs.[6]

COMMENT

Despite this patient's extreme sensitivity to oral medication, intravenous guanethidine required 48 hours to effect reduction in the blood pressure. The differences in recumbent and standing blood pressures in this case were considerable. Syncope occurred at least a dozen times, each time without any warning symptoms of dizziness, weakness or sweating. A scalp laceration was sustained during one of these episodes.

Extreme sensitivity to guanethidine was also reflected by the diarrhea produced by small doses of this drug. The patient did not show any reduction in blood pressure on moderate doses of bretylium tosylate, indicating that no undue sensitivity to this agent was present in this case.

The increased sensitivity to guanethidine in this patient may in part have been due to the decrease in renal function and renal excretion of the drug.

OTHER PATIENTS TREATED

Ten other patients received guanethidine for two to 16 months, as the sole agent for treatment of their hypertension. The side effects have been minimal. The pre-treatment and post-treatment blood pressures in these cases are listed in Table II. Case 1 had symptoms of postural hypotension in the morning after a single evening dose of guanethidine. These symptoms disappeared when the daily dose was divided. Slight dizziness occurred in the patient of Case 2 after heavy manual exertion. Impotence was another side effect experienced by this patient. In Case 5 the blood urea value was 150 mg. %, and as renal function deteriorated further the severe hypertension was controlled by less than 10 mg. daily. The response in Case 6 has not been entirely satisfactory, but attempts to increase the dose of guanethidine were followed by the appearance of postural symptoms. Another patient (Case 7) developed some tolerance to guanethidine, as evidenced by recurrence of pre-treatment blood pressure levels after an initial favourable response, but a satisfactory result was obtained by doubling the dose. In Cases 6 and 10 there was an initial response to bretylium, but tolerance developed and increasing the dose did not produce a satisfactory result.

DISCUSSION

In the evaluation of any drug for antihypertensive therapy it is important that the drug should be used alone. Only under these conditions can its effects be determined with accuracy. Furthermore, though combinations of antihypertensive drugs may

TABLE II.—BLOOD PRESSURE RESPONSE TO GUANETHIDINE

Case	Age	Initial B.P. lying	B.P. standing	On guanethidine therapy Lying B.P.	Standing B.P.	Daily dose (mg.)	Side effects
1	68	200/120	190/120	160/100	140/90	25	Occasional dizziness
2	20	160/100	160/110	140/90	130/80	50	Occasional dizziness, diarrhea
3	12	160/100	160/100	140/90	120/70	12.5	Nil
4	46	172/110	190/110	130/90	120/85	30	Nil
5	40	220/130	210/140	180/115	124/76	25	Nil
6	52	270/150	260/140	200/120	160/110	75	Morning dizziness
7	54	220/110	210/94	195/88	130/60	50	Nil
8	56	200/112	210/112	190/90	140/88	20	Nil
9	13	170/110	160/110	130/75	110/68	25	Occasional loose stool
10	56	240/130	200/140	160/80	130/70	75	Occasional dizziness

occasionally be beneficial, therapy should be kept as simple and as inexpensive as possible, since it may be required for a lifetime.

Because of its minimal side effects guanethidine has a distinct advantage over all of the ganglion-blocking agents, but it still constitutes a symptomatic form of management of the hypertensive patient, and it may fail to lower the supine blood pressure sufficiently in some cases. Adequate control of supine blood pressure in malignant hypertension leads to a better prognosis than that of patients whose standing blood pressures alone are reduced.[8] Guanethidine did not significantly reduce peripheral resistance in hypertensive patients studied by Richardson *et al.*[10] Its mode of action at clinically tolerable doses consists mainly of decreasing the venous return to the heart and the cardiac output, and in this regard is similar to pentolinium.[11] Thus the pathological physiology of hypertension, increased peripheral vascular resistance, is not necessarily corrected.

Page and Dustan[9] have emphasized that tolerance may develop to bretylium tosylate, and the failure of large doses of this drug to lower the blood pressure in the two patients with malignant hypertension described in this report would suggest that guanethidine may be a more effective antihypertensive drug. Also, tolerance developed in two other patients who initially responded to bretylium tosylate.

SUMMARY

Preliminary experience with guanethidine in the treatment of hypertension has been reported.

Observations of its effects on 12 hypertensive patients indicate that guanethidine is a potent hypotensive agent.

Sensitivity to this drug may vary greatly from patient to patient. One of two patients with malignant hypertension required 800 mg. for effective blood pressure reduction; the other patient required only 25 mg. daily.

Postural hypotension and syncope may occur without warning.

Two to three days are required for a hypotensive effect even when guanethidine is given intravenously. Its hypotensive action persists for two days or more after the drug has been discontinued.

The sensitivity of individual patients to guanethidine and to bretylium tosylate varies, suggesting that these drugs may have a different mode of action. For two patients with severe hypertension, guanethidine was effective in lowering the blood pressure, while bretylium tosylate was not.

Guanethidine used in this study was supplied by Ciba Company, Dorval, Quebec, through Drs. W. Murphy and C. A. Schaffenburger. Bretylium tosylate was supplied by E. Scrapneck of Burroughs Wellcome Company. The skin temperature studies were performed by Dr. P. Gaskell of the University of Manitoba. Dr. J. P. Gemmell reviewed the manuscript.

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PAGES OUT OF THE PAST: FROM THE JOURNAL OF FIFTY YEARS AGO

And yet someone may ask: Is alcoholism not closely associated with degeneracy? To that I would answer: Yes, but as a symptom or a result, and not a cause, of degeneracy. I hold that to regard it as a cause of degeneracy is about as futile as to claim that intellectual deficiency is the cause of the defectively developed brain.

Nor can racial degeneration be brought about by underfeeding any more than by overfeeding. You will hear it not infrequently stated that the economic conditions of our modern civilization are responsible for the degeneracy of type which may be found in the slum districts of densely populated cities, and that if economic conditions were improved not only would misery and poverty disappear, but degeneracy would be eliminated from the race. Misery and poverty may be alleviated in this way, but to hope that degeneracy would thus vanish is to indulge in a foolish dream. Can the economist, the philanthropist, or the statesman, or all three combined, add by thinking on their behalf one cubit to the intellectual stature of a family of mental degenerates? In the words of Sidney Smith, "You might as well try to poultice away the humps of a camel."—A. B. MacCallum: The Ancient Foundations of Heredity, *Canadian Medical Association Journal*, 1: 3, January 1911.

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FURTHER OUTBREAKS OF BOTULISM IN CANADA*

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IN THE SIX YEARS since authenticated occurrences of botulism in Canada were last reviewed in this journal,[1] a further ten outbreaks have been investigated. All of them took place in British Columbia or Labrador, and uncooked marine products were implicated in every instance.

Pacific Coast Indians were involved in three outbreaks due to salmon eggs, while Eskimos at the other extreme of the country were the victims on six occasions in which seal flippers, meat or liver were the botulinic vehicles. These food-stuffs were in various stages of decomposition, so that most Canadians would consider them rather disgusting to look upon, and impossible to eat. However, some of the native inhabitants of our coastal perimeter continue to expose themselves to an endemic botulism hazard whose control requires that it be given wide publicity.

An earlier report covering the period 1919-53 listed 14 occurrences, including 3 type A due to home-bottled vegetables, and 3 type E due to home-canned or pickled fish.[1] Thus, over the last 40 years, there have been 24 recorded outbreaks involving 87 persons, with 50 deaths—a case fatality rate of 57.5%. In 18 outbreaks, or two-thirds of the total, the implicated foodstuffs were inadequately cooked or pickled fish, raw fish eggs prepared unsanitarily, or products of sea mammals, especially the seal and the white whale. Home-preserved vegetables were responsible on six occasions, and home-canned meats twice.

A few comments seem pertinent on the incidence of the disease, the kinds of foods concerned, and the types of organisms involved, in Canada as compared with certain other countries. In the United States, according to data compiled by Meyer and Eddie,[7] during the 50-year period 1899-1949 there were 477 reported outbreaks of botulism affecting 1281 persons, of whom 830 died, i.e. a case fatality rate of 65%, slightly greater than the corresponding Canadian figure. After due allowance is made for differences in total population and in the periods covered by the available statistics, the calculated incidence of outbreaks and the morbidity rate are also somewhat higher in the United States. But the disparity is now far less than in 1947 when attention was originally drawn to it in a report on the first occurrence to be diagnosed bacteriologically in Canada—the type E outbreak in 1944 at Nanaimo, B.C.[8] At that time, only three previous episodes of human botulism had been recorded in this country. If present trends continue, the position as regards incidence may be reversed; for in the United States, but not in Canada, reported outbreaks of botulism appear to be declining

More significant differences are apparent between these two countries in respect of the foodstuffs involved and the types of *Cl. botulinum* identified. In the United States, canned vegetables and fruits have caused 75%, but in Canada only 25%, of reported outbreaks. On the other hand, fish and marine products were responsible for 66.7% of Canadian occurrences, but for only 5.7% of the United States total. In both countries, about 8% of all episodes were traced to meat products—

TABLE I.—HUMAN BOTULISM IN CANADA (1954-60)

Date	*Place of occurrence*	*Cases*	*Deaths*	*Foodstuffs implicated*	*Type*
1954	Bella Bella, B.C.	3	1	Salmon egg "cheese"	E
1956	Ungadlek, Labrador	8	6	Uncooked seal flipper	E
1957	Prince Rupert, B.C.	3	3	Uncooked salmon eggs	E
1958	Port Edward, B.C.	1	1	Uncooked salmon eggs	B
1960	Penticton, B.C.	1	0	Salted Holland herring	E
1960	Hopedale, Labrador	4	2	Uncooked seal flippers	?
1960	Hopedale, Labrador	1	1	Seal liver	A
1960	Nr. Hopedale, Labrador	1	1	Dried seal meat	?
1960	Nr. Hopedale, Labrador	1	0	Dried seal meat	?
1960	Nain, Labrador	1	0	Uncooked seal flippers	?

Total outbreaks, 10; cases, 24; deaths, 15; case fatality rate, 62.5%.

In the tenth episode, an elderly woman, residing in an inland city of southern British Columbia, had a moderately severe attack of the disease after consuming salted herring imported from Holland. As this case presents certain unusual features, the relevant clinical, epidemiological and laboratory data will be outlined.

INCIDENCE AND EPIDEMIOLOGY

The outbreaks of botulism recognized throughout Canada since 1953 are summarized in Table I. Of 24 persons affected, 15 died. Samples of the suspected foods yielded type E strains of *Clostridium botulinum* in four instances, while types A and B strains were each isolated on single occasions. The first four outbreaks listed have been reported previously,[2-5] and the unique sequence of five Labrador episodes in the summer of 1960 will also be described elsewhere.[6]

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TABLE II.—FOODSTUFFS CAUSING BOTULISM IN THE UNITED STATES AND CANADA EXPRESSED AS PERCENTAGES OF TOTAL OUTBREAKS, CASES AND DEATHS

	Vegetables and fruits			*Preserved and canned meats*			*Fish, shellfish and marine mammals*			*Totals**		
	O	*C*	*D*	*O*	*C*	*D*	*O*	*C*	*D*	*O*	*C*	*D*
U.S.A.	75.0	80.2	81.5	7.7	7.3	7.2	5.7	5.2	4.6	88.4	92.7	93.3
Canada	25.0	38.0	34.0	8.3	6.9	2.0	66.7	55.1	64.0	100	100	100

O = outbreaks; *C* = cases; *D* = deaths.

*The totals for the United States fall short of 100% because in some instances the vehicle was unknown or unclassifiable.

the customary agents in Germany and France (Table II).

The numbers of types A, B and E human botulism outbreaks identified, and the total case fatality rates, in Canada, the United States, and several other countries, are set forth in Table III. In Canada, the 12 strains of *Cl. botulinum* isolated from 50% of the total outbreaks, comprised four type A, one type B and seven type E. By contrast, in 149 outbreaks, or 31.2% of the total cited for the United States,[7] type identification showed the following distribution: Type A, 119; B, 25; both A and B, 2; and E, 3. None of the three type E occurrences was due to food of local origin. In fact, the earliest of them, which took place at Cooperstown, N.Y., in 1932, and retrospectively proved to have been the world's first known type E outbreak, was due to smoked salmon from Labrador.[9] Although in the last decade there have been six more type E occurrences, all in Alaska (*vide infra*), their inclusion in any up-to-date revision of Meyer and Eddie's figures would not appreciably diminish the striking overall preponderance of type A botulism.

pigs were clandestinely slaughtered, and hams improperly salted, smoked or pickled at home, at least 500 outbreaks of botulism occurred, involving over 1000 cases, with a fatality rate of less than 2%. Porcine foodstuffs containing type B toxin were responsible for 93% of all episodes. Type B strains were predominantly involved in the remaining 7% of outbreaks, which were due to home-preserved non-porcine meats, fish and vegetables; but a few of these, including one episode with three fatalities, traced to tuna fish soup, were caused by type A strains.[15] About 10 years ago, three fish-borne episodes, involving five persons, none of whom died, were attributed to type E toxin, but without conclusive proof.[16]

If comparisons be made with countries whose fish consumption is relatively high, striking differences in type distribution and other epidemiological features are again apparent. For instance, in the United Kingdom, botulism was unknown until 1922, when eight persons died in the well-known type A outbreak at Loch Maree, Scotland, after eating wild duck paste sandwiches. Since then, there have been six other outbreaks, the vehicles

TABLE III.—TYPES OF CL. BOTULINUM IMPLICATED AND CASE FATALITY RATES FOR BOTULISM IN VARIOUS COUNTRIES

Country	*Period covered*	*Recorded outbreaks*	*Type identifications*			*Untyped*	*Case fatality rate (%)*
			A	*B*	*E*		
Canada	Up to 1960	24	4	1	7	12	57.5
United States	1899–1949	477	121*	27*	3	328	65.0
France	1940–44	500	3	202	—	295	1.5
United Kingdom	Up to 1960	7	3	1	—	3	76.5
Norway	Up to 1960	10	—	7	—	3	Nil
Denmark	Up to 1958	10	1	—	1	7†	40.6
Japan	1951–60	33	—	—	33	—	31.9

*Includes two outbreaks in which types A and B strains were both isolated.
†An outbreak due to the new type F strain accounts for the apparent discrepancy.

Except for Japan, Canada is the only country in which type E outbreaks predominate. For example, although the prototype E strains were isolated from sturgeon in the Soviet Union in 1934-35, very little botulism of any type has been reported from there since then. Presumably this is largely due to the imposition of rigorous sanitary controls over the fishing industry during the past quarter-century. Formerly, when fish-borne botulism was acknowledged as a serious public health problem in parts of Russia, type A outbreaks outnumbered type B.[14] In France, on the other hand, type B outbreaks are far commoner than type A. During the 1940-45 occupation, when meat was scarce, including rabbit and pigeon broth, jugged hare, meat pie, and a vegetarian dish known as "nut meat brawn". Pickled fish was incriminated in one type A occurrence, in 1955, but the fish was caught and prepared four weeks before in Mauritius, where no botulism has ever been recognized.[17] Altogether, there have been seven outbreaks, with 17 cases and 13 deaths, showing a remarkably high fatality rate of 76.5%. Of the four bacteriologically identified outbreaks, three were type A, and one type B.

In Norway, 7 out of 10 known outbreaks of botulism have been due to home-canned or salted and dried meats, chiefly ham. Home-canned fish

cakes were implicated once, while salted fish, although very popular, was responsible for only two episodes, which occurred inland and involved single individuals. The vehicle was "rakefisk", a Scandinavian dish in which mountain lake trout are compressed between layers of salt in a wooden tub, and stored at around 15° C. The NaCl content of the brine was about 5%. Type B toxin was demonstrated in the causal foodstuffs of seven outbreaks, including the two fish-borne ones. No deaths were reported among the total of 51 persons affected.[18, 19]

Between 1901 and 1958, Denmark likewise experienced 10 outbreaks of botulism, involving 32 persons, of whom 13 are known to have died—a case fatality rate of some 40%. These differed in several respects from the Norwegian and British outbreaks. One-half of the occurrences were due to fish products, salted or pickled mackerel and herring being implicated four times, and commercially canned salmon once. Preserved liver loaf was responsible twice, while on three occasions the causal foodstuffs were unknown.[20] There were no type B isolations in this series, but one outbreak (vehicle unspecified) was shown to be type A. Another outbreak, due to herrings in oil, was identified as type E. As a sequel to this latter episode, the presence of type E organisms was demonstrated in mud specimens taken from the bottom of Copenhagen fish canal and harbour.[21] The latest occurrence, on the island of Langeland in 1958, affected four persons, one of whom died, and was due to home-made liver loaf from which a strain of *Cl. botulinum* of an apparently new type was isolated in the State Serum Institute, Copenhagen.[22] Investigations of this strain in the author's laboratory indicate the propriety of designating it a prototype F culture.

The predominance in Canada of type E botulism, and of fish and marine mammals as vehicles, are interrelated phenomena. This fact is best illustrated by the situation in Japan, where botulism went unrecognized until 1951. Since then, in Hokkaido and northwestern Honshu – the less populated northern regions of the country—there have been 33 outbreaks, all type E, affecting 188 persons, of whom 60 died. With only one exception (involving canned mackerel) these outbreaks have been due to "izushi", a locally popular relish comprised of fermented rice, dried vegetables and raw fish. Type E organisms have been isolated from dead fish, from sea shore sand and lagoon mud, and to a lesser extent from nearby soil, in the endemic areas. There can be little doubt that locally caught, bottom-feeding fish, or their predator species, are the botulogenic components of "izushi", and that these become contaminated with type E spores either endogenously through their intestinal tracts, or exogenously through their integuments. Comestibles containing such fish, if kept for some time before consumption and then eaten raw, are very dangerous.[6, 10]

Analogous observations and principles fully account for the verified outbreaks—two type E and one type B—due to salmon eggs, among coastal Indians of northern British Columbia,[2, 5] for botulinus spores (nearly always type E) have been demonstrated in sea bottom samples taken off the coast of this province at depths of from 6 inches to 658 metres. Since 1948, there have been at least five similar outbreaks—two of them identified in this laboratory as type E—involving 7 Indians, with 6 fatalities, in neighbouring southeastern Alaska.[6, 11] Various techniques are used by Indians of this region in the preparation of fish eggs for consumption. In the 1940 outbreak in the Yukon, which was the earliest recorded occurrence of this kind, ripening of the eggs was accelerated, and an extra fillip given to their flavour, by keeping them stuffed in a moose bladder for ten days.[5] In this instance, the potentialities for botulinic contamination were particularly extensive. Other methods range in complexity from rather lengthy desiccation rituals to mere suspension of the eggs in a jar of their own juice, where they mature for 7 to 10 days in the open air. The simpler methods provide fewer opportunities for soil contamination, direct or indirect, so that any botulinus spores present are more likely to have originated in the intestinal contents of the fish. But as the mode of preparation becomes more elaborate, opportunities multiply for telluric contamination. In the 1954 outbreak at Bella Bella, B.C., for example, chum salmon eggs were placed in a gunny sack, washed in a fresh water stream, and then spread out in the smoke house. After being smoked for several hours, the eggs were squeezed by hand into a firm mass and packed into a wooden crate to ferment for at least two weeks in the open air, being poked with a stick now and again to allow gas to escape. The cured mass was then transferred to tins for further maturation.[2]

In the light of findings in northern Japan and the northwest Pacific coast region of North America, and to a small extent in Denmark, it seems justifiable to conclude that type E botulism is most liable to occur in areas where dietetic customs entail consumption of uncooked products of fish and marine mammals, *provided that* type E spores be fairly heavily disseminated in terraqueous deposits of the local littoral. There are two corollaries to this conclusion: (1) in areas where type E outbreaks are endemic, type E spores should be demonstrable in samples of sea bottom mud or of shore-line sand collected in the vicinity, and (2) if this form of the disease does not occur in a given region despite predisposing dietetic habits, type E spores are either sparsely distributed or non-existent in the locality.[6] The latter set of circumstances presumably obtains in the British Isles and northwestern Europe—except for the known focus at Copenhagen—while the former seems likely to be exemplified by the Eskimos of Alaska, the Northwest Territories, and Labrador.

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Although no surveys for *Clostridium botulinum* have yet been made of sea bottom or shore samples taken from the arctic and subarctic periphery of the continent, specimens are now being collected from several areas for this purpose, and it may be conjectured that type E spores will prove to be focally present and probably predominant over other types. Since the 1945 tragedy at Markham Bay, N.W.T., in which 7 Eskimos died out of 8 made ill by eating raw or parboiled seal meat,[12] there have been 12 additional outbreaks among North American Eskimos, involving 36 persons, of whom 16 died. Six of these occurred between Point Hope and Scammon Bay along the coast of northwestern Alaska from 1947 to 1959, the vehicles being raw beluga flippers preserved in seal oil ("muktuk") in five instances, and the fluke of a decomposing gray whale in the sixth episode.[6] Specimens of the implicated food were procured on four occasions, and each time yielded a type E strain of *Cl. botulinum*.

In the remaining six outbreaks, which took place betwen Nain and Hopedale on the Labrador coast, 24 Eskimos were affected, with 17 fatalities. These were all due to uncooked foodstuffs prepared from seals, and with one exception, developed in a period of a few weeks from the middle of May to early July 1960. In two occurrences at or near Nain, and one at Hopedale, the vehicle was "utjak", or seal flippers kept in seal oil, usually in a cask near the stove, until suitably rotten. The local Eskimos blame these tragedies upon replacement of wooden tubs by metal containers for storing the flippers. There may be an element of truth in this claim in that metal absorbs and conducts more heat, but unofficial records of whole families found dead in isolated areas, and stories told by Eskimos of their parents or grandparents having died of "bad seal meat",[13] suggest that botulism has been endemic in this area for some time. Two recent outbreaks near Hopedale, involving dried seal meat as vehicle, occurred among a community of Eskimos who had emigrated the year before from a much colder district 250 miles farther north, where it was customary to cut seal steaks of unusual thickness for drying. The centre of these steaks remained far from dry, and was liable to putrefaction, and in the relatively warm summer climate of Hopedale presumably furnished suitable conditions for botulinus toxin production.[13]

In the foregoing occurrences, the sources of botulinus spore contamination of the seal flipper or meat could have been either endogenous (intestinal) or exogenous (telluric). The route of contamination in the fifth episode is less equivocal. In this instance, a Hopedale woman died of typical acute botulism after eating some raw seal liver, from a carcass that had lain intact under a porch for 3½ days after being caught. As the victim herself cut the liver out just before consuming it, the route of contamination could hardly have been through damaged integuments. The organisms are much more likely to have reached the liver from the seal's intestine, via the portal system, either before death or agonally. In this favourable medium, the prevailing mid-summer temperatures would permit rapid proliferation and toxin production. A small portion of the seal liver, forwarded deep frozen to this laboratory, contained no detectable toxin, but a highly toxigenic type A strain of *Cl. botulinum* was isolated from it. Nothing significant could be cultured from samples of stomach washings forwarded in two instances, or of liver taken at autopsy from one of the victims in the first of the 1960 outbreaks.

Before last summer's series of outbreaks, the only authenticated occurrence in Labrador took place at Ungadlek, near Nain, in 1956. Six persons died of 8 affected as a result of eating "utjak". In this instance, seal flippers coated with seal oil had been allowed to rot for 10 days at the bottom of a dirty gasoline drum, close to a stove at an estimated temperature range of 20-40° C.[4] One of the remaining flippers, sent to this laboratory, contained type E toxin, and yielded a type E culture.[3]

SYMPTOMATOLOGY

In all the foregoing episodes the symptomatology was unmistakable, and need not have presented diagnostic problems to physicians aware of the endemic botulism hazard confronting Indians and Eskimos of the Canadian North. In general terms, in the areas cited, a diagnosis of botulism is justified when a previously well person develops a rapidly progressive, afebrile illness featuring abdominal disturbances, general weakness and malaise, plus evidence of cranial nerve palsies, within 8-20 hours after consuming uncooked products of fish or sea mammals.

The following description of the acute botulinic syndrome, as manifested among the natives of arctic and subarctic regions of Canada (and Alaska), is based on accounts of 11 outbreaks involving 43 Canadian Eskimos and Indians, of whom 33 died, a case fatality rate of 76.7%. The onset is usually insidious but relentless, with nausea, vomiting, abdominal pain and distension. The constipation which is so characteristic of the later stages of the disease may be preceded by a diarrheal phase, possibly due to heavy contamination of the foodstuff with other bacteria. The often quoted allegation that conspicuous vomiting usually presages a fatal outcome was not borne out in this series. (Vomiting was common to most cases, and may have contributed to recovery in a few instances by ridding the stomach of some unabsorbed toxin.) The clinical diagnosis is clinched when these gastrointestinal symptoms are accompanied or superseded by complaints of abnormalities of vision, difficulty in swallowing, alteration of voice, and dryness of mouth. In fulminating cases, increasing weakness culminates in a state of total collapse, as the paralytic process affects the limbs and the

circulatory and respiratory centres. The pulse becomes feeble and the blood pressure critically low, while dyspnea and deepening cyanosis give place to frank air hunger, and the hitherto conscious patient lapses into a brief terminal coma.

In several cases in this series, death occurred in less than 24 hours, and there were no fatalities later than the third day after the toxic food was eaten. The generally rapid course no doubt reflects the high proportion of outbreaks due to type E toxin, which acts on the whole more quickly than types A and B toxins. Also, the outcome in type E cases is especially unpredictable, probably because type E toxin is liable to be activated in the upper gastrointestinal tract on encountering trypsin at certain pH levels (5.5-6.5), so that variable amounts of additional toxin are released.[10] Type B toxin is less subject, and type A toxin not at all, to this process. In the first and largest outbreak of botulism in Canada, at Dawson, Y.T.,[23] uninvestigated bacteriologically, the minimum incubation period among 23 affected miners was 26 hours. The first death occurred on the third day after commercially canned beets were eaten[24] and 11 others died in the next 8 days. These facts alone rule out type E toxin, and point to type A, as the source.

The high case fatality rate prevailing in these outbreaks among Indians and Eskimos underlines the serious botulogenic hazards in such foodstuffs as salmon eggs, "muktuk" and "utjak". It also suggests that less serious cases are going unreported. The following account of a moderately severe, sporadic occurrence in a small city where skilled medical attention was available, emphasizes how easily mild forms of botulism could pass unrecognized anywhere. Since this is the first recorded instance of salted fish being implicated in type E botulism, the possible modes of contamination and times of toxin production will be discussed.

CASE REPORT

At the end of February 1960, a 64-year-old housewife living at Penticton, B.C., purchased a small barrel of salted herring from a local food store. She kept the keg in the refrigerator for 10 to 14 days, when she opened it, washed the fish overnight to free them of salt, and put them in a dish with vinegar. The following day she ate approximately two small herring in a salad at a late evening meal. Her husband, who took very little of the fish, suffered no ill effects, but the woman awoke next morning feeling rather weak. Soon after a breakfast of bacon and eggs, she developed left-sided abdominal pain and vomited.

A few hours later, she was seen by a physician, who found her pale, somewhat weak, with a blood pressure of 190/100 mm. Hg, and a tender left abdomen, especially the upper quadrant. She was given 100 mg. of meperidine, and her husband was told to report if the pain and vomiting recurred. At this stage, the alternative diagnoses were considered to be diverticulitis, acute gastritis, small bowel obstruction, or possibly even a coronary thrombosis. After two hours, the husband telephoned that there was no improvement, whereupon the patient was admitted to hospital, some 24 hours after the meal. She now complained of dizziness in addition to general weakness and left-sided abdominal discomfort, but seemed otherwise in fair condition. A white blood cell count, an electrocardiogram, and a flat x-ray plate of the abdomen were ordered for the next morning. However, about 12 hours after admission, before any of these procedures had been carried out, she suddenly became much weaker, and experienced difficulty in swallowing, breathing and talking. She was pale and sweating, with blood pressure 140/90 and pulse rate 72. Botulism was first suspected at this juncture. The electrocardiogram (ECG) showed nothing abnormal. The radiological examination revealed apparent paralysis of one loop of small bowel, which spread to a large segment before disappearing—an observation interpreted as due to absorption of toxin mainly from this area. Two weeks after the illness began, the patient had recovered, except for generalized fatigue and numbness of the left hand showing ulnar nerve distribution, which persisted for several weeks.

A few comments bearing on this clinical history will be interjected. Late relapses and delayed deaths may occur in type E as in other types of botulism. For example, in one episode in Alaska, an Eskimo woman suddenly died on the ninth day of illness, after she had apparently begun to recover;[6] and in the interior of British Columbia a man died 18 days after eating home-pickled river trout.[12] Perhaps such occurrences are due, as certain Russian authorities contend,[25] to botulism of any type being actually a "toxinfection", in which there is some degree of invasion of the host's viscera with production of toxin *in situ*. But an acute exacerbation of symptoms at an earlier stage, as noted here, seems especially characteristic of type E botulism, and could result from the already mentioned process of tryptic activation of the toxin to higher lethal potentialities. Conditions appropriate for this process are likely to obtain over a relatively short length of the upper intestine. The x-ray findings suggest that the zone of maximum absorption of toxin (which does not necessarily correspond to the zone of activation) may also be of restricted extent. The radiological characteristics of botulinic intestinal and esophageal paralyses have been stressed particularly in France,[26] and should be more widely known, as should bradycardia, extrasystoles, and other abnormalities of conduction which may be present in botulism,[15, 20] but were not observed in this instance. Finally, in the light of the following laboratory findings, the patient appears fortunate not to have suffered a more severe or even fatal attack, instead of escaping with minor and transient sequelae.

A sample of the remaining herring, amounting to about two fish, was shipped to this laboratory for examination. The specimen was moist and friable, but not grossly abnormal in appearance or odour. A Gram-stained preparation showed heavy contamination with mixed cocci and bacilli. Some Gram-positive rods, many with oval, subterminal

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spores, were noted (Fig. 1). A portion of the fish was macerated and extracted in physiological saline overnight in the refrigerator, and centrifuged. The presence of type E botulinus toxin was demonstrated in the supernatant to the extent of roughly 600 mouse minimal lethal doses (MLD) per gram of fish. Using techniques previously described,[3] a type E strain of *Cl. botulinum* was isolated from the herring, which produced 1000-3000 mouse MLD of toxin per ml. of laboratory culture medium.

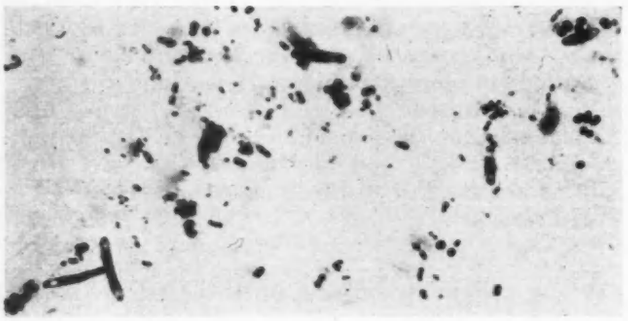

Fig. 1.—Gram-stained smear of suspension of salted herring, implicated in Penticton case of type E botulism. Note several dividing bacilli, with subterminal oval spores. (× 1200.)

Assuming that this 175-lb. woman consumed an estimated 400 g. of herring, and that the toxicity of the sample did not significantly change before being titrated in the laboratory, she would have ingested about 240,000 mouse MLD of type E toxin. This amount is almost identical with the calculated 250,000 mouse MLD of toxin taken by an Indian woman (weight 150 lb.) who recovered from a very severe attack of botulism in the salmon egg outbreak at Bella Bella, B.C.: and is about one-half the amount of toxin consumed by a sister (weight 180 lb.), who died within 24 hours of eating 6 tablespoonfuls, or about 90 g., of the eggs.[2] Actually, if high degrees of *in vivo* activation of toxin occur, such as were demonstrated in the Prince Rupert outbreak,[5] far lesser amounts of ingested toxin than the above may prove fatal. In that episode, one of the victims who died claimed to have eaten no more than two teaspoonfuls (around 10 g.) of salmon eggs containing about 250 mouse MLD of type E toxin per g., i.e. her ingested dose was only 2500 mouse MLD.

Two questions of epidemiological interest arise: (1) Did the type E spores gain access to the fish from marine or terrestrial sources? (2) At what stage between netting of the fish in the North Sea, and their consumption in Penticton some 8000 miles away, did the spores vegetate, with subsequent proliferation and production of toxin?

The keg had contained 6½ lb., or about 3 kg., of "milker" (male) herring packed in strong brine (35% NaCl). British Columbia herring at present cannot be sold profitably for human food, but there is a consumer market for an imported product from Holland, where a salted herring industry has thrived for centuries, based on catches netted over a wide area of the North Sea. On board the boats, the whole fish are packed in large barrels between layers of salt, after their intestines have been pulled out through a nick near the gills—a rapid but clumsy cleaning procedure which facilitates pollution of large numbers of herring with bacterial spores of fish intestinal origin. On land, the barrels are held in refrigerated storage pending export, before which the fish are sorted by sex and transferred to coopered kegs, each keg of "milkers" containing 12-15 male herring. While in transit, and on arrival at their destination, such kegs are again refrigerated until distributed to retailers, who do not necessarily store them in the cold. In this instance, the implicated keg had been shipped to the retail store about one month before it was purchased, where it was kept alternately in the meat counter display case, and in a walk-in cooler, at temperatures a few degrees above freezing point.

The presence of botulinus spores, especially type E, in sea mud and fish intestinal samples from certain regions of the northern hemisphere, has been established as one of the main reasons why type E botulism is almost exclusively conveyed by fish and sea mammals.[6, 10] The methods of harvesting and packing these salted herring obviously provide opportunities for pollution of a keg's contents with spores of fish fecal origin. But since this is the first occurrence of botulism attributed to fish caught in the North Sea, presumably these waters, unlike certain areas of the northern Pacific Ocean, carry very few type E spores.[6] Alternative modes of contamination to be considered in this episode are inward seepage of spores through cracks in the keg during storage, and pollution from soil while the fish were being desalted prior to consumption. The latter possibility is strengthened by the demonstrated presence of type E spores in British Columbia soil.[8] The actual source of the spores in this instance cannot be settled.

The time of toxin production likewise remains uncertain, although its elaboration in frozen, heavily salted herring can be ruled out.[27] The low temperature and the high NaCl concentration could help to preserve any preformed toxin present in a relatively stale fish introduced accidentally into the barrel before addition of the salt. But any such toxin would probably be washed away, along with most of the salt, when the fish were soaked in water overnight. A final opportunity for toxin production was afforded by the 24-36 hour interval between desalting and consumption of the fish. Our laboratory data indicate that it would have been possible, in the circumstances then prevailing, for some type E strains to manufacture significant amounts of toxin in the herring. The conditions, though by no means optimal for type E toxin production, were no more unpropitious than those prevailing in the 1951 pickled herring episode at Vancouver.[27]

DIAGNOSIS

Despite its almost pathognomonic symptomatology, acute botulism is liable to be confused with various infective and toxemic conditions, of which the following are the most important: poliomyelitis, encephalitis, diphtheria, and tick paralysis; tetraodon and paralytic shellfish poisoning; trichinosis; atropine, carbon monoxide, and methyl alcohol poisoning. Before discussing the salient clinical differences between these conditions, it may not be superfluous to stress that simple common sense will often set aside some of the alternatives. For example, neither arthropod-borne encephalitis nor tick paralysis need be considered in arctic regions where the requisite vectors do not exist; ichthyotoxism and paralytic shellfish poisoning likewise have restricted geographical distributions. Again, botulism is more likely than atropine poisoning in a Labrador Eskimo, or than methanol poisoning in an orthodox Doukhobor living in the southern interior of British Columbia. Further, a few questions relating to foodstuffs eaten on the preceding day or two can often furnish a decisive clue.

The absence of fever in all but the terminal stages of acute botulism is a crucial point of differentiation from bulbar poliomyelitis and the encephalitides. Also, in both the latter conditions, cerebrospinal fluid abnormalities are present; and in a severe encephalitic attack, the mentality is at least clouded. Diphtheria should be distinguishable by the presence of a membranous inflammatory focus, by the absence of abdominal disturbances such as constipation and meteorism, and by differences in paralytic phenomena. The ptosis and diplopia so common in botulism are rare in diphtheria, while tendon reflexes often lost in the latter disease may persist in the former. Tick paralysis is a relevant consideration only in regions where the North American wood tick, *Dermacentor andersoni*, is abundant. In Canada, this hemophagous vector, and the peculiar paralytic disease it may cause in sheep, cattle and occasionally man, are chiefly confined to the dry belt of British Columbia.[28] The paralysis is typically ascending, the feet and legs being first affected, resulting in a staggering gait. Although dysphagia may develop before respiratory paralysis supervenes, ocular signs and symptoms are rare. Fever and a rapid pulse are usually present. Removal of the tick in time usually leads to fairly prompt recovery.

Tetraodon or puffer fish poisoning has mainly a circumtropical distribution and is unknown in Canada. But it is a serious health problem in Japan, where no doubt it has been confused sometimes with fish-borne (type E) botulism. Sensory disturbances, which are absent in botulism, are conspicuous in this condition. Numbness and tingling generally begin in the lips and tongue within 20-45 minutes after ingesting puffer-like fishes, spread to the extremities, and may eventually involve the whole body. Increasing malaise, ataxia and respiratory distress accompany these paresthesias. Hypersalivation, profuse sweating and sometimes extensive petechial hemorrhage are other distinguishing features. In paralytic shellfish poisoning, a very similar syndrome (but without petechiae) develops soon after consumption of clams, mussels, or oysters containing an alkaloid present in certain species of Gonyaulax—a dinoflagellate plankton which periodically swarms in ocean waters off the Pacific and Atlantic coasts of Canada.[29] The nature of the food consumed, the short incubation period and the presence of sensory disturbances should suffice to rule out botulism.

Trichinosis, despite its protean manifestations, shows few close resemblances to botulism, and ought never to be confused with it. In both the intestinal and invasive stages of trichinosis, fever is present; the incubation period before the onset of abdominal symptoms is several days; and the phase of muscle pains, facial swellings and rash lasts for some weeks. It is included here because both diseases are endemic among the Eskimos of North America and result from consumption of raw or inadequately cooked flesh of Arctic mammals; and also because the explorer Stefansson, who in 1914 correctly surmised the existence of trichinosis in the Arctic, later wrongly attributed to this disease certain mysterious fatalities which wiped out whole groups of Eskimos who had eaten white whale meat.[6]

Among chemical intoxications simulating botulism in certain respects, atropine poisoning is nowadays a rare contingency, mentioned chiefly because it is traditionally quoted in this connection. Doses of atropine large enough to cause toxic effects beyond ocular paralyses and suppression of secretions would generally induce psychic disturbances, which do not occur in botulism. In severe carbon monoxide poisoning, there is likely to be evidence of recent occupational, accidental or suicidal exposure. Abdominal symptoms are inconspicuous, the patient's colour is pinkish or ashen instead of cyanotic, and he is either stuporous, unconscious or in a state of mental confusion. Finally, methyl alcohol may cause some diagnostic difficulties when there is no immediate history that wood alcohol has been ingested, especially if consumption of a possibly botulogenic food can be verified. A case of this type occurred a few years ago in Nova Scotia. A seriously ill woman with ocular paralyses, dyspnea and convulsions became comatose and expired 23 hours after eating home-bottled baked beans. Subsequently, a high level of methyl alcohol was demonstrated in her blood and brain, while the beans showed no evidence of contamination by *Cl. botulinum*.[30] In acute methanol poisoning, disturbances of vision may be accompanied by pain and tenderness in the eyeballs; and manifestations of cerebral excitation, such as convulsions, hallucinations and even maniacal behaviour, often precede the onset of coma.

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Subacute attacks of botulism, and their sequelae, are especially liable to be mistaken for conditions ranging from "a bout of 'flu", "a touch of polio", and "neurasthenia", to toxic myocarditis and sundry causes of bowel obstruction and paralytic ileus. In mild and ambulant cases, which are probably fairly common in some countries, including Canada, the correct diagnosis is unlikely to be thought of by the average physician, except perhaps in Germany and France. Wherever the disease is an endemic hazard, the peculiar combination of persistent lassitude or weakness, constipation, fuzziness of vision or difficulty in accommodation, and perhaps slight dysphagia, should arouse suspicions of a botulinic etiology.

Verification of a clinical diagnosis of botulism involves demonstrating the presence in the implicated foodstuffs, or in the vomitus or stomach contents of the victim, of a heat-labile toxin, whose lethality for experimental animals is neutralizable by monovalent botulinus antitoxin, generally of type A, B or E. The isolation from such toxic specimens of an homologous strain of *Cl. botulinum* is important confirmatory evidence; and when the samples submitted happen to be atoxic, but yield a toxigenic culture, the finding can still be regarded usually as conclusive. The techniques in such investigations have been described elsewhere.[3, 5] Here it must suffice to stress the importance of shipping promptly to the laboratory any available samples of suspected foodstuffs, however stale and fragmentary. They should be sent by air express in previously sterilized specimen containers or household sealers, preferably packed in dry ice. Over the past 15 years, in every human botulism occurrence in Canada and Alaska from which a remnant or companion sample of the suspected food was forwarded to this laboratory, a culture of *Cl. botulinum* has been obtained. Of 17 strains thus isolated, 4 were type A, 1 type B, and 12 type E. Some samples of most unpromising appearance gave fruitful results. For instance, a tiny piece of herring backbone, covered with coffee grounds, retrieved from a garbage can where it had lain for three days, yielded a type E strain;[27] and a type A strain was isolated from corn cobs which had been thrown from the kitchen doorway into the snow over two weeks before.[1]

If none of the suspected food be left, the house should be searched for canned, bottled or otherwise preserved foods of the same batch, and any found should be forwarded to the laboratory. Often all relevant food samples are destroyed, in which event less satisfactory but still worthwhile types of specimens may be collected. Toxin is frequently demonstrable in vomitus or gastric contents taken at autopsy and small amounts are sometimes detectable in the blood; it may be possible to culture the causal organisms from vomitus, gastric contents (and even stomach washings), feces, and the spleen or liver of fatal cases.

TREATMENT

Many handicaps must be surmounted in the treatment of botulism, and few therapeutic situations can be more frustrating. In its more acute forms, the disease seems to progress inexorably. Moreover, the diagnosis is usually made retrospectively after the patient is dead, moribund, or at least gravely ill; and yet it cannot be confirmed for at least 24 hours after the laboratory has received a sample of the suspected food. Finally, whether or not the type of toxin responsible can be identified promptly, trivalent (types A, B and E) botulinus antitoxin is at present impossible to procure commercially in North America.

Botulinus toxin, after being absorbed from the gastrointestinal tract, is carried to sites adjacent to cranial and peripheral motor nerve endings, where it exerts an inhibitory effect upon the mechanism of acetylcholine synthesis or release, thus interfering with the transmission of neural impulses to muscles and glands. The main therapeutic objectives are therefore: (1) to evacuate or render inert as much toxin as possible, before its absorption from the stomach and intestines; (2) to neutralize circulating toxin in transit to susceptible sites; (3) to counteract, or compensate for, the action of the toxin upon neuromuscular junctions and neurosecretory mechanisms. In attaining each of these objectives, type specific or polyvalent botulinus antitoxin could play a vital part. It is deplorable that such materials are not (with minor exceptions) available from strategically situated depots across Canada: they are in the United Kingdom, despite the far lower incidence of botulism there. In the Soviet Union, on the basis of animal experiments, it is advocated that antitoxin be administered perorally, intragastrically or intraduodenally, as well as by the more conventional route.[25] In France, for many years, botulism has been treated by combining repeated intramuscular injections of antitoxin with subcutaneous doses of toxoid, using polyvalent products until the type concerned is known, and homologous ones thereafter.[15] Ambulant cases, and botulinic sequelae, are also said to have responded favourably to immunization by homologous toxoid.[16] Such toxoids are relatively simple to prepare, but are not commercially available for human use in North America, although type C toxoids for prevention of mink botulism have a brisk market. Recently, single intramuscular injections of type E antitoxin prepared by the Connaught Medical Research Laboratories, in co-operation with the Defence Research Board of Canada, have given very encouraging results in two severe type E cases in Hokkaido, Japan,[31] and in one bacteriologically unidentified case due to seal meat in Labrador.[13]

Supplementary measures range from rigorous stomach lavage and high enemas to the use of a mechanical respirator. The stomach washing is advisedly carried out with an alkaline solution,

such as 2 to 5% sodium bicarbonate in water. This serves the triple purpose of ridding the stomach of considerable toxin, of exposing residual toxin to alkaline pH levels at which the degeneration rate is high, and of minimizing the tryptic activation process which affects especially type E toxin, but only within pH limits of 5.5 to 6.5. In severe cases, a mechanical respirator should be resorted to early, to forestall respiratory paralysis. Such appliances, suitable for transport by air, should be kept in readiness for this purpose at all outpost hospitals in the Canadian North.

Symptomatic and supportive treatment are important, but drugs such as pilocarpine, strychnine and acetylcholine, which theoretically should be useful, seem to give disappointing results. Administration of antibiotics is pointless, except when bronchopneumonic complications threaten to supervene.

PREVENTION

The high thermal stability of type A botulinus spores is still not widely recognized as a hazard inherent in home-preserved vegetables and fruits grown on soil containing these spores, as in the Grand Forks area of southern British Columbia.[1] Although it is in fact impossible to guarantee destruction of all type A spores in a given bottle subjected to ordinary home-preserving treatment, type E spores, which cause most botulinic troubles in Canada, are relatively so heat-labile that raw or slightly heated foods are the commoner menace from this standpoint. If our native Indians and Eskimos would boil their salmon eggs, "muktuk", and "utjak" for a few minutes, or even heat them at 80° C. for 20 minutes, any type E spores present would almost certainly be destroyed. However, under prevailing social and environmental conditions, there is a great risk that recontamination of such foods might occur during storage, causing them to become unsafe again by the time they were ready for consumption. Therefore some other preventive measure must be sought.

Since type E strains can multiply and produce toxin at the surprisingly low temperature of 6° C.,[27] and have an optimal temperature range for growth and toxin production 5 to 10° C. lower than that of types A and B strains,[32] it is futile to rely upon storage of these marine foods in a relatively cool place. Moreover, smoking, drying, pickling and even strong salting of fish products are obviously no safeguard against botulism. The soundest prophylactic measure, regardless of the potential vehicle and the type of toxin involved, is thorough exposure of the food to a temperature of 80° C. for 20 minutes, or to 90° C. for 10 minutes, within an hour or two of its consumption. Such foods cannot convey the disease, because all botulinus toxins are thus destroyed. This simple fact should be stressed in public health educational campaigns designed to reduce the dangers from botulogenic foods. If the native populations of Canada, who are particularly concerned, cannot or will not follow this advice, efforts should be made to wean them (especially the children and younger parents) from their traditional but insanitary and dangerous diets. Acculturation will accomplish this purpose eventually, but too slowly to prevent the needless loss of many more lives.

SUMMARY

In the past six years, 10 outbreaks of botulism have occurred in Canada, involving 24 persons, of whom 15 died. In all instances, uncooked products of fish and marine mammals were the vehicles. Coastal Indians in British Columbia were affected in three instances, while on six occasions (five of them in the summer of 1960) the victims were Labrador Eskimos. The tenth episode involved a woman in Penticton, B.C., who recovered from type E botulism due to salted herring imported from Holland; this case has been described in detail.

Over a 40-year period, 24 outbreaks of botulism have been recorded in Canada, affecting 87 persons, with 50 deaths, i.e. a case fatality rate of 57.5%. The implicated foodstuffs in two-thirds of these occurrences have been fish and marine mammals. The coastal regions of British Columbia and Labrador have been the setting for a great majority of all outbreaks.

Cl. botulinum has been isolated by one laboratory from remnant or companion foodstuffs in 12 instances. The strains were identified as four type A, one type B and seven type E. Comparisons are made between the type distribution, the kinds of foods concerned and the case fatality rates, in Canada and several other countries.

The epidemiology, symptomatology, differential diagnosis, treatment and prevention of botulism are reviewed, special consideration being given to its status as an established endemic hazard in certain parts of Canada.

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ISOLATION OF ENTERIC VIRUSES DURING THE POLIOMYELITIS SEASON IN ONTARIO, 1956-1959*

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THE PURPOSE of this communication is to acquaint the practising physician with the prevalence and epidemiology of various enteric viruses in Ontario and with the variation in their respective preponderance from year to year.

The term "poliomyelitis season" as used in this communication is defined as the period from June 1 to November 30 of each year. This period was chosen because only an insignificant number of isolations of enteric viruses was made during the rest of the year. During four such seasons 3700 specimens were examined from which 1128 virus isolations were made. Whenever possible the etiological significance of the isolates was verified by serological methods.

EPIDEMIOLOGICAL FEATURES

Poliomyelitis

Poliomyelitis occurred to an epidemic degree in 1956 and 1959.

In 1956, specimens from 133 paralytic cases, 13 fatal, were submitted for study. The outbreak in June occurred in Northern Ontario and was of Type I. Later in the season there were sporadic cases of all three types all over the province. In addition specimens from 73 "non-paralytic" poliomyelitis cases were also examined (Table I).

In 1957, specimens from only 35 paralytic cases, 8 of them fatal, were received and in 1958, specimens from 23 cases, 3 of them fatal. These specimens were from sporadic cases from all parts of Ontario.

In 1959, specimens from 200 paralytic, 8 fatal and 27 non-paralytic cases were submitted for study. Outbreaks started simultaneously in various parts of Ontario. Most of the isolations were of Type I. The ages of patients ranged from 6 months to 57 years, but 67% fell into the "below-14 year" age group; 45% were under 5 years of age.

Aseptic Meningitis

This syndrome was widespread in all four years, though the entiological agents (Poliovirus I, ECHO 2, 5, 6, 7 and 9, Coxsackie A9 and Coxsackie B 1-5) varied from year to year. The condition known in 1956 and earlier as "non-paralytic poliomyelitis" should properly be included here. Outbreaks of this syndrome occurred simultaneously in many parts of Ontario. It affected mostly the young adult population (20-35 years), although the actual ages ranged from the newborn to 80 years.

Epidemic Pleurodynia and Epidemic Pericarditis

In 1958, in addition to the above syndrome, a number of the population suffered from epidemic pleurodynia and epidemic pericarditis. Forty-five cases of pleurodynia and 25 of pericarditis were proved to be of Coxsackie B5 etiology, but many more cases were reported in various parts of Ontario. The epidemic started in mid-July and reached its peak in September. The ages of the patients ranged from 5 to 78 years, but the majority fell into the 20-35 year age-group.

MATERIALS AND METHODS

Specimens

Specimens examined included feces, cerebrospinal fluids, throat washings, pleural and pericardial aspirations, bloods and organs obtained at autopsy. About half of the out-of-town specimens received were frozen immediately after collection and shipped in dry ice to Toronto. Local specimens, as a rule, were delivered to the laboratory within 24 hours after collection.

A 1/10 suspension was prepared from all solid specimens in Earle's solution containing 500 units/ml. of penicillin, 100 μg./ml. of streptomycin and 100 units/ml. of mycostatin.

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Tissue Cultures

Roller tube cultures of trypsinized monkey kidney and HeLa cells were routinely employed. The monkey kidney tissue cultures were grown in Medium H 597[1] and the HeLa cells in Eagle's medium.

If specimens of special interest failed to produce a cytopathogenic effect in these cell cultures, they were in addition inoculated into human amnion, human embryonic skin-muscle, human foreskin and human adult heart tissue cultures.

Inoculation of Suckling Mice

All specimens failing to show a cytopathogenic effect in tissue cultures were routinely inoculated intraperitoneally and intracerebrally into day-old mice. A representative number of the viruses isolated in tissue cultures were also passaged in mice to study the lesions produced. Histological sections were made of the brain, skeletal muscles, fat pads and various organs. Hematoxylin-eosin staining was used throughout.

Specific Sera

Specific antisera for the three types of poliovirus, Coxsackie B types 1-5, Coxsackie A types 1-19, ECHO types 1-20 and Adenoviruses types 1-10 were prepared in rabbits.*

Serology

The neutralization test, employing both microscopic and colorimetric methods, was used for the typing of isolates and the titration of antibodies in the patients' sera. All tests were made using 100 $TCID_{50}$/0.1 ml. of the specific virus. The typing antisera contained 20 or more antibody units per 0.1 ml. The patients' sera were tested at twofold dilutions from 1:4 to 1:1024.

Paired sera—acute and convalescent—whenever available, were titrated simultaneously, but a considerable number of single convalescent sera was also examined. There was no specific time at which specimens were collected, and for our purpose an arbitrary division of acute and convalescent specimens was set at five days after onset of symptoms.

RESULTS

Seasonal distribution of the viral isolations over the four years is recorded in Fig. 1.

In each season the earliest isolations were made at the beginning of June. In 1956, the isolations were most numerous in September and then gradually declined during October and November. The greatest number of isolations made during 1957 were in August, with a sharp decline in October and ending by December. In the 1958 season, the peak of isolations shifted to October, with an abrupt decline in November. In 1959, most of the isolations were made in September, although a considerable number were recovered both in August and October. Although the greatest number of viral isolations were made during the same three-month period each year, the types of viruses varied. As can be seen from Table II, in 1956 both poliovirus and ECHO 9 were the most prevalent. In 1957, ECHO 9 predominated, while in 1958 the majority of the isolates belonged to the Coxsackie group, almost all of them being Coxsackie B type 5. In 1959, once again Poliovirus type I was predominant, but there was also a large number of Coxsackie B type 2 and type 5 isolations. Coxsackie B type 2 was isolated for the first time in the four-year study and accounted for just over 10% of total isolations in 1959.

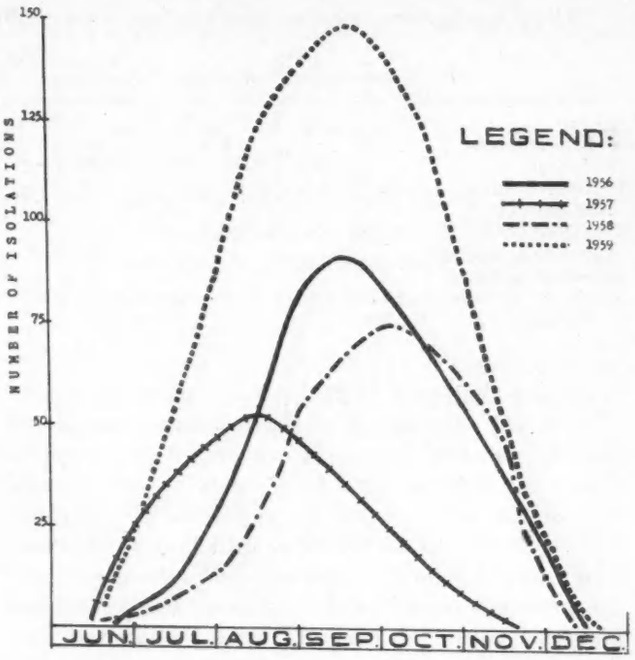

Fig. 1

The distribution of viruses isolated from the 3700 specimens examined during the four-year period, with respect to the nature of the specimens, is shown in Table III. The greatest number of isolations were from feces specimens, followed by acute phase blood, cerebrospinal fluid (CSF), autopsy material and throat washings. The small number of isolations from throat washings is understandable, as most of the specimens were collected well after the clinical syndrome was established. The large proportion of isolations from blood may be somewhat misleading, as only selected specimens taken within 72 hours after onset of earliest symptoms were examined.

Poliovirus

In 1956, specimens from 206 cases clinically diagnosed as poliomyelitis were submitted for examination (Table I). Poliovirus was isolated from 111 of them, representing 39% of the total virus isola-

*Some of the antisera were kindly supplied by the Dominion Virus Laboratories, Ottawa.

TABLE I.—ISOLATION OF POLIOVIRUS IN THE YEARS 1956, 1957, 1958 AND 1959 FROM CLINICAL CASES OF POLIOMYELITIS

	1956		*1957*		*1958*		*1959*	
	Specimens	*Isolations*	*Specimens*	*Isolations*	*Specimens*	*Isolations*	*Specimens*	*Isolations*
Fatal poliomyelitis...	13	4	8	4	3	2	8	6
Paralytic poliomyelitis...	120	97	27	15	20	13	192	124
Non-paralytic poliomyelitis...	73	10						
Aseptic meningitis			8	2			27	4
Healthy contacts.					26	13		
Total.....	206	111	43	21	49	28	227	134

tions for that year. Of these, 81 were of type I (Table II). Almost all of the isolations were from frankly paralytic cases. However, 10 isolations of Poliovirus type I were from cases which showed only slight and transient or no paresis. No information with regard to previous infection or vaccination was available in these non-paralytic cases, and no sera were submitted for antibody studies. Of the paralytic cases, 71 had not been vaccinated and 5 had received two or more doses of poliomyelitis vaccine (Salk type); for the other 44, no information was available.

In 1958, 23 cases of paralytic poliomyelitis, 3 fatal, were investigated and Poliovirus type I was isolated from 15 of them (Table I). Only two of the 23 cases had had poliomyelitis vaccine. In 1958, in addition, the feces of 26 healthy persons who had one member of the family ill with Poliovirus type I yielded Poliovirus type I in 13 instances. Four of the persons had "flu" or mild gastric upset at about the same time. All except one had received two or more doses of poliomyelitis vaccine.

TABLE II.—ISOLATIONS OF ENTEROVIRUSES IN THE YEARS 1956, 1957, 1958 AND 1959

Year	*Total isolates*	*Poliovirus*			*Coxsackie*						*ECHO*						*Not identified*
		I	*II*	*III*	*A9*	*B1*	*B2*	*B3*	*B4*	*B5*	*1*	*2*	*5*	*6*	*7*	*9*	
1956	288	81	21	9	0	0	0	6	4	5	—	—	—	—	—	139	23
		39%			0			4.5%					48%				8%
1957	152	18	2	1	3	2	0	5	1	3	—	—	—	—	—	101	16
		14%			2%			7%					66%				11%
1958	223	28	0	0	6	1	0	4	2	136	—	—	—	—	—	17	29
		12.5%			2.5%			64%					7.5%				13%
1959	465	127	0	7	11	0	47	4	15	65	1	6	1	6	1	17	157
		29%			2%			28%					6.5%				34.5%

NOTE: — = not tested.

In 1957, from 35 poliomyelitis cases, 8 of which were fatal, 19 isolations were made (Table I). All except 3 were of Poliovirus type I (Table II). In addition, Poliovirus type I was isolated from two cases of transient paresis which were clinically diagnosed as aseptic meningitis (Table IV). Of the 35 cases, 21 had not received any poliomyelitis vaccine, two had two doses of vaccine, and no information was available for the remaining 12.

In 1959, 200 specimens from paralytic cases, 8 fatal, were examined. Poliovirus type I was isolated from 125 cases (including 6 fatal cases) and Poliovirus type III from 5 cases. From 27 specimens of patients with aseptic meningitis, Poliovirus type I and type III were each isolated from two cases. A surprising isolation was of Poliovirus type I from

TABLE III.—TYPES OF SPECIMENS EXAMINED AND VIRUSES ISOLATED

Type of specimens examined	*Number examined*	*Viruses isolated*					*Isolations*	
		Poliovirus	*Coxsackie*		*ECHO*	*Not identified*	*Total*	*Percentage*
			A	*B*				
Stools.....	2431	290	18	241	254	204	1007	41.4%
Throat washings.....	325	1	0	10	1	3	15	4.6%
CSF.....	830	1	2	41	28	15	87	10.5%
Acute phase blood.....	40	0	0	8	4	1	13	32.5%
Autopsy material.....	74	2	0	0	2	2	6	8.1%
Total.....	3700	294	20	300	289	225	1128	30.5%

CSF of a paralytic case. (Careful investigation has ruled out laboratory contamination.)

The longest time interval between the onset of paralysis and the recovery of a virus was four weeks, the greatest number of isolations being made in the first two weeks of the illness. Feces from fatal cases could not always be obtained. From those which were available, isolations could be made in only half of them. From the 32 samples of feces from fatal cases, 11 isolations of Poliovirus type I, two of type II and three of type III were made. In only two fatal cases the brain and spinal cord were made available for study and in both cases Poliovirus type I was recovered.

blood specimens taken within 48 hours of the onset of illness.

Although all the strains were typed serologically as ECHO 9, they fell into two categories when inoculated into day-old mice.

(a) 66% were found to be pathogenic for infant mice and produced a widespread Zenker-type degeneration of striated muscle. There were no changes in the brain, fat pads or various organs examined.

(b) The remaining 34% of the strains failed to produce any lesions in the infant mice and resembled the prototype Hill strain.[2]

TABLE IV.—VIRUS ISOLATIONS FROM CASES OF ASEPTIC MENINGITIS, 1956 - 1959

	Poliovirus			*Coxsackie*						*ECHO*					*Not identified*
	I	*II*	*III*	*A9*	*B1*	*B2*	*B3*	*B4*	*B5*	*2*	*5*	*6*	*7*	*9*	
1956.....	10	—	—	—	—	—	6	4	5					139	
1957.....	2	—	—	3	2	—	5	1	3					101	7
1958.....	—	—	—	5	1	—	4	2	57					17	8
1959.....	2	—	2	5	—	43	3	15	57	6	1	6	1	17	27

Paired sera, whenever available, were examined for a rise in neutralizing antibody titre. Unfortunately the number of such specimens submitted was small. However, in the unvaccinated paralytic cases a four-fold or greater increase in titre to the specific virus type was clearly seen. In the few vaccinated cases, the picture was variable and unclear.

ECHO 9 Virus

In 1956, a large number of the specimens from which ECHO 9 virus was isolated was submitted from cases clinically diagnosed as "non-paralytic poliomyelitis". By 1957, however, the term "aseptic meningitis" largely replaced "non-paralytic poliomyelitis", referring to a clinical diagnosis without etiological implications. In 1956, 48% (139 strains) of the total isolations were ECHO 9 (Table II). In 1957, the percentage rose to 66% (101 strains) and ECHO 9 was isolated from specimens submitted from practically all parts of Ontario. A remarkable reversal of the trend was shown in 1958, when only 7.5% (17 strains) of the total isolations were of ECHO 9. In 1959, ECHO 9 represented only 3.5% of the total isolates. In addition, ECHO types 2, 5, 6 and 7 were isolated in small numbers from cases of aseptic meningitis (Table IV). One isolation of ECHO type I was made from a liver specimen obtained at autopsy where death was sudden and cause unknown. No other significant bacterial or viral isolations were made from this case.

Again, feces specimens yielded the greatest number of viruses, and some isolations were made as late as four weeks after the onset of symptoms. On the other hand, isolations from CSF and throat washings were made only during the first ten days of the illness. Viremia was proved in four cases and the isolations were made from acute phase

Both types were found in each of the eight Ontario cities and towns which were studied. Serological response of patients who had exhibited clinical symptoms and yielded a virus was uniformly rather poor. Of the paired sera tested, 9 had a convalescent titre between 1:4 and 1:32 and only one showed a rise in titre to 1:128.

Coxsackie Viruses

In 1956 and 1957, isolations of Coxsackie viruses were very few, totalling 26 for two years. The distribution according to type is shown in Table II. In 1958 the picture was quite different. Of the 223 total virus isolations 143, or 64.5%, were of Coxsackie viruses, nearly all being Coxsackie B type 5.

Coxsackie A viruses, other than type A9, could be isolated only in suckling mice and these were isolated from cases presenting vague symptoms of pyrexia, sore throat, malaise and occasionally aching muscles. No further typing of these viruses was attempted. Isolations of Coxsackie A9 were made in tissue culture, all but one being isolated from cases of aseptic meningitis. The exception was from a case of pyrexia and aching muscles.

All primary isolations of Coxsackie B viruses, with one exception, were made in tissue culture. The exception, a Coxsackie B5 strain from a case of pleurodynia, grew readily in tissue culture only after primary mouse passage.

Coxsackie B types 1, 3 and 4 were isolated sporadically each year from cases of aseptic meningitis (Table IV).

Coxsackie B type 2 appeared for the first time in 1959 and in considerable numbers. It was also isolated mostly from cases of aseptic meningitis, but in addition, it was recovered from four cases of pleurodynia.

TABLE V.—ISOLATIONS OF COXSACKIE VIRUSES IN THE YEARS 1956, 1957, 1958 AND 1959

	1956						1957					
Coxsackie	*A9*	*B1*	*B2*	*B3*	*B4*	*B5*	*A9*	*B1*	*B2*	*B3*	*B4*	*B5*
Non-paralytic poliomyelitis..	0	0	0	6	4	5	2	2	0	3	1	3
Aseptic meningitis.....	0	0	0	0	0	0	1	0	0	2	0	0
	1958						1959					
Coxsackie	*A9*	*B1*	*B2*	*B3*	*B4*	*B5*	*A9*	*B1*	*B2*	*B3*	*B4*	*B5*
Aseptic meningitis.....	5	1	0	4	2	57	8	0	43	3	15	57
Pleurodynia.....	0	0	0	0	0	45	0	0	4	1	0	6
Pericarditis.....	0	0	0	0	0	25	0	0	0	0	0	2
Pneumonia.....	0	0	0	0	0	0	2	0	0	0	0	0
Pyrexia of unknown origin...	1	0	0	0	0	9	1	0	0	0	0	0

Coxsackie B type 5 was seen only occasionally before 1958. However, in 1958 and 1959 it was isolated in large numbers. In 1958, 57 isolations of Coxsackie B type 5 were from cases of aseptic meningitis, 45 from pleurodynia, 25 from pericarditis and 9 from cases of pyrexia of unknown origin and abdominal pain (Table V).

In 1959, the greater majority of Coxsackie group B isolations were from cases of aseptic meningitis. Only 11 isolations were made from 40 cases of pleurodynia. Of these, four were of Coxsackie B type 2, one of Coxsackie B type 3 and six of Coxsackie B type 5. Only two isolations of Coxsackie B type 5 were made from 29 specimens submitted from cases of acute idiopathic pericarditis.

Paired sera in many cases were examined for the presence of neutralizing antibodies. As a rule, high titres were observed in the specimens collected within the first week of illness. Usually it was only when the acute blood was taken sufficiently early (2-3 days after onset of illness) that a four-fold or greater increase in titre to the homologous virus could be demonstrated. High titres, consistently greater than 1:512 in both phase sera (when the acute phase blood was taken after the third day), have been considered as evidence of a recent infection.

The results of the isolations from cases of aseptic meningitis for the four-year study have been recorded in Table IV.

Dual Isolations

In four cases of aseptic meningitis laboratory findings were rather surprising. In the first case, ECHO 9 was isolated from the acute blood specimen and Coxsackie B type 5 from the feces. In the second case, ECHO 9 was isolated from the blood and Coxsackie B type 5 from the CSF and the feces. In the third case, ECHO 9 was isolated from the feces and Coxsackie B type 5 from the CSF. In the fourth case, ECHO 9 was isolated from the CSF and ECHO 9 and Poliovirus type I from the feces (Table VI). All specimens from each patient were obtained on the same day. Second feces specimens examined four weeks later yielded a Coxsackie B type 5 virus only in the second case.

Two phase sera were available only in the first and fourth cases. In the first case the antibody titres to Coxsackie B type 5 were 1:32 and 1:64 in the blood samples taken on the 5th and 15th day of the illness respectively, but no antibody titre to ECHO 9 virus in either specimen was demonstrable. In the last case antibody titre to ECHO 9 virus was 1:32 in the first and 1:128 in the second serum specimen. No antibody titre to Poliovirus type I was demonstrable in either of the two serum samples, and no antibodies to either virus could be demonstrated in the acute phase sera of the other two cases.

Not Identified Viruses

The term "not identified viruses" as used in this communication refers to the isolates which were not neutralized by the antisera for Poliovirus types I, II and III, Coxsackie type A9, types B 1-5, and ECHO types 1, 2, 3, 4, 5, 6, 7, 8, 9, 12 and 16. These isolations were made from specimens submitted from such cases as were diagnosed as pyrexia of unknown origin, "flu", aseptic meningitis and non-paralytic poliomyelitis.

TABLE VI.—DUAL ISOLATIONS

	Virus isolation				*Serum titre*		
	Feces						
Aseptic meningitis cases	*Specimen 1*	*Specimen 2*	*CSF*	*Blood*		*Acute phase*	*Convalescent phase*
Number 1.....	Coxsackie B5			ECHO 9	B5	1:32	1:64
					E9	—	—
Number 2.....	Coxsackie B5	Coxsackie B5	Coxsackie B5	ECHO 9	B5	—	not
					E9	—	available
Number 3.....	ECHO 9	—	Coxsackie B5		B5	—	not
					E9	—	available
Number 4.....	ECHO 9		ECHO 9		E9	1:32	1:128
	Polio I				Polio I	—	—

A particularly large number of these "not identified viruses" were recovered in 1959. Their identification is still in progress.

COMMENTS

During the four-year study, virus isolations were made from about a third of the specimens submitted. This number of isolations could probably have been increased if appropriate specimens had been taken earlier in the disease and submitted in better condition.[3] The necessity of early sampling also applies to the collection of acute phase serum, especially in the case of Coxsackie infections where high neutralizing antibody titres are developed within the first week of the disease.

Approximately 90% of the total isolations were made from feces specimens. This, however, does not indicate that other specimens should be neglected. Much remains to be learned about enteroviruses, and all possible sources of material should be studied. Multiple specimens and two-phase sera especially are necessary if the etiological significance of each of the multiple viruses isolated from the same patient is to be determined.

In this study it was observed that, although the great majority of poliovirus isolations were from paralytic cases, a few were from cases presenting only meningismus or some vague symptoms.

Of particular interest was the isolation of Poliovirus type I from the CSF of one paralytic case of poliomyelitis. The CSF had been collected on the second day of the clinical illness. The patient had not been previously vaccinated. Repeated isolations under carefully controlled conditions several months apart from the same specimen (which was kept frozen) ruled out laboratory contamination.

It was also noted that poliovirus and ECHO 9 infections to epidemic degrees did coexist in 1956 (Table II). The incidence of the poliomyelitis and Coxsackie group of viruses in the same season to an epidemic degree could not be evaluated clearly. However, in 1959, Poliovirus type I represented 28% of total isolations, while Coxsackie B type 2 (10%) and Coxsackie B type 5 (14%) totalled 24%.

The aseptic meningitis syndrome of ECHO 9 or Coxsackie B type 5 etiology was more disturbing than dangerous, death or permanent paresis being almost non-existent in our experience. In only one case might ECHO 9 have been the cause of death. A seven-day-old boy died within two hours of the onset of respiratory difficulties. Autopsy revealed marked interstitial pneumonitis, and bacteriological examination failed to show significant organisms. Monkey kidney tissue culture inoculated with the specimens of blood and lung showed the presence of ECHO 9 virus. Suckling mice inoculated with the original specimens showed marked striated muscle degeneration.

Coxsackie B type 5 strains were isolated from patients with a multiplicity of syndromes. Some of the cases were clear-cut in symptomatology and presented only one clinical picture, either that of aseptic meningitis, pleurodynia, pericarditis or pyrexia with abdominal pain. However, a considerable number presented overlapping pictures with one of the syndromes predominating. In a few, there were relapses with the same or a different clinical syndrome.

The mixed infections observed in four cases of aseptic meningitis are difficult to evaluate by the data at hand. A virus isolated from the CSF or blood is considered to be the cause of the infection. The simultaneous presence of another virus in feces may or may not be significant. However, the simultaneous presence of one virus in the spinal fluid and another in the blood cannot be accounted for satisfactorily. Cross contamination in the laboratory has been ruled out. In all four cases recovery was prompt, and in none were there symptomatic relapses suggestive of two separate illnesses quickly following one another. It is unfortunate that the serological data which might have helped in understanding these cases were not complete.

SUMMARY

A review of the various enterovirus isolations during the years 1956, 1957, 1958 and 1959 is presented.

In 1956, paralytic and non-paralytic poliomyelitis were the main clinical pictures. From a total of 288 isolations, 111 were of poliovirus and 139 of ECHO 9.

In 1957, aseptic meningitis of ECHO 9 etiology was the only epidemic form, totalling 101 of the 152 isolations made in that season.

In 1958, an epidemic of Coxsackie B type 5 infection occurred with various manifestations. Of a total of 223 isolated viruses, 136 were of Coxsackie B type 5; 57 of these were from cases of aseptic meningitis, 45 from cases of pleurodynia, 25 from cases of pericarditis and 9 from cases of pyrexia with severe abdominal pain.

In 1959, there was an epidemic of Poliovirus type I and 127 isolations were made from 227 specimens submitted. In addition, a considerable number of Coxsackie B type 2 viruses were isolated from cases of aseptic meningitis for the first time in four years. Eleven isolations of Coxsackie B types 2, 3 and 5 were made from 60 cases of pleurodynia and 2 of Coxsackie B type 5 from cases of acute idiopathic pericarditis.

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DEVIANT SEXUAL BEHAVIOUR AND THE SEX CRIMINAL

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WHILE IT IS TRUE that the average man in our Western civilization spends a great deal of his waking life fantasying and ruminating over sexual matters, it is difficult to estimate just what proportion of his time this occupies. It is likely true that the time spent thinking about such matters is markedly disproportionate to the time spent in real sexual activity. It is also true that one of the striking differences between the commission of a crime and the restraining of oneself from such commission is merely the difference between thinking a thought and acting out a thought. The inhibiting effect of a social and moral conscience is usually the differentiating factor. If the thinking about criminal deeds was equally as much a crime as the commission of such deeds, then we would likely all be criminals at one or other time in our lives.

Where social and moral conscience is weak or absent, a psychopathic or sociopathic personality is usually in existence. Or to put it in a simpler manner, whereas a normal person respects and is aware of the difference between right and wrong, so too is a psychopath often aware of social wrong, except that he does not *care* about it. He is thus able to carry out antisocial behaviour without the pangs of conscience that most "normal" persons would experience. Where such activity involves sexual behaviour, he may become a sex criminal (but such criminals are not necessarily all psychopathic personalities). This will also depend upon whether or not he is discovered by the law, or apprehended by the law. This raises a perplexing question. Does a crime occur whenever behaviour described as criminal by a criminal code takes place, or only if law enforcement bodies know of it? For the answer to this, we must ask help from our legal colleagues.

The element of conscience need not be involved in a so-called sex crime, however. Even if this action follows mutual consent between two adult homosexuals and neither calls upon the law to intercede, the action can still be considered criminal if a legal officer learns of it. In some states in the U.S.A., various deviant sex practices between a man and a woman are crimes punishable by law even with mutual consent between the two, should the law be informed.

The increasing overt interest in our civilization regarding sexual matters in the past few years (as witnessed by the popularity of certain novels, plus the sexual themes of the majority of successful motion pictures) is commonly accepted, but not without concern, by many. It is difficult to believe that such public interest was not as marked in the Victorian era, although of course in a more subdued manner. So often, "wrong", "guilt", "sin", and similar terms are applied to sex, and being a "good" or "bad" person involves one's handling of one's sexual activities, thoughts and interests.

A combination of public concern over sexual matters in general, plus mounting interest in the causation of sex crimes, disposal and rehabilitation of the criminal population and the sex criminal in particular, has led to the writing of this paper.

What follows below combines the author's experience in the treatment of patients with sex deviations (with some personal reflections therefrom), plus gleanings from the reports of colleagues and other authors who have dealt with similar patients in recent years.

The public reaction to sex deviation of any sort—always stronger when children are involved—is one of alarm, resentment, and disgust, and with varied prejudice against the one who commits the deed. For years now, the public trend has been turning to the realization that such persons are ill—socially if not medically—and that prison alone is not the answer to their problem.

The inflammatory reaction of such deeds upon the public may produce temporary chaos and emotional turmoil, but may often bring about a worthwhile outcome, as happened in the city of Toronto six years ago. Three Toronto housewives, as a result of their concern over several sex crimes involving children in that city in 1954-55, banded together to found the "Parents' Action League", a lay organization dedicated to the pursuit and dissemination of knowledge about sex crimes, and research into treatment and possible prevention of such phenomena. An impressive list of psychiatrists, medical men, psychologists, legal personnel, politicians and lay persons were involved in the creation of the body, which received much public support, and support from the lay press. As a result of its efforts, and pressure directed by it upon the Ontario Provincial Government, the "Forensic Clinic"—an affiliate of the Toronto Psychiatric Hospital and the University of Toronto Department of Psychiatry—was born. The clinic was not entirely oriented to sex deviates even at first, although interest was predominantly in sexual deviation at that time. The Parents' Action League[1] branched out, as a lay organization, to concern itself also with juvenile delinquency, salacious literature, education in the schools regarding accepting rides from strangers, and other pertinent areas of concern. Chapters formed throughout the province, and interest in the organization spread through the nation and to the U.S.A.

Why do (usually) men commit sex crimes, and what are the most common types? The breakdown of statistics for new cases seen in 1958 from the Forensic Clinic in Toronto[2] indicates that the most common types of antisocial sexual behaviour are

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homosexuality, exhibitionism, and pedophilia. Of 179 new patients seen in 1958, only six were suffering from psychoneurotic disorders and 13 were schizophrenic; the majority of the remainder were persons with disorders of character, behaviour and intelligence, but not necessarily victims of mental or emotional illness as we know it.

Table I indicates the frequency of the more common types of sex crime in the city of Edmonton (population about 250,000) for the years 1957-59 inclusive, as described by the Edmonton City Police.

TABLE I.

Sex crimes as described by the city of Edmonton police department	*1957*		*1958*		*1959*	
	Occurrences reported	*Arrests or summonses*	*Occurrences reported*	*Arrests or summonses*	*Occurrences reported*	*Arrests or summonses*
(a) Exhibitionism and indecent acts (other than urinating on street, etc.)	52	18	68	15	83	15
(b) Rape	8	6	16	10	15	18*
(c) Sexual assaults involving female children	18	10	25	16	20	12
(d) Homosexual acts involving adults	3	4	6	6	4	2
(e) Homosexual offences involving children	10	6	8	4	7	5
(f) Bestiality	2	1	2	1	0	0

*More than one male involved in some of these offences and therefore multiple arrests.

Hammer and Glueck[3] studied 200 male sex offenders at Sing Sing Prison, under a program sponsored by the New York State Department of Mental Hygiene, over a period of five years, and concluded that sex offenders have *castration fears* (but not necessarily consciously) in the following order of increasing intensity, according to the crime committed: (1) rape, (2) heterosexual contact with adolescents and children, (3) homosexual actions with adolescent partners, and (4) homosexual action with child partners. [*Castration fears* refers to the conscious or subconscious anxiety experienced by men with reference to loss of, or threats to their masculinity, virility, physical appearance, etc. Men with such a problem often have difficulty relating easily to women, feel inferior, and so forth.] The continuum, from rapist to homosexual pedophilia, appears to represent in parallel fashion the increasing intensity of castration feelings on the one hand, and the correspondingly greater distance from the mature female as a potential sexual object on the other. Incest subjects were found to be the most overtly psychotic, and also to harbour the most intense castration feelings. The view held by these authors is that the sex offender presents an attempt on the part of the offender to employ *substitute* sex outlets for the mature female, because of the threatening potential, both psychosexually and psychosocially, with which she is endowed. Almost every one of the subjects exhibited feelings of fear (as a reaction to massive Oedipal entanglements) of approaching *mature* females psychosexually.

As in the case of obesity, hopes for endocrinological causation of sex deviation are legion, but rarely realized in fact. So also is the case in physical treatment of sexual aberration, especially with the use of hormonal substances. One study[4] has shown that sex offenders have a low average 24-hour 17-ketosteroid excretion, though not statistically significant. A recent report[5] from the Verdun Protestant Hospital in Montreal showed that stilbestrol was of definite value in suppressing undesirable and socially uninhibited sexual behaviour in only six of the 15 chronic mental hospital patients on whom it was tried. These could well be persons without the finer attributes of personality functioning, and thus personally unbiased subjects for such research.

Castration of sexual offenders is permitted by law in many countries, notably Norway.[6] If it is effective, likely its value is due to the psychic trauma and fear inflicted, and not to specific physiological or psychological treatment as such. This practice in some ways resembles that of some ancient civilizations, which amputated the hand of a criminal because of theft.

Lieberman and Siegel[7] reported on a program for the treatment of sex offenders at a state hospital in California over a period of two years from 1952 to 1954. In 284 patients involved, 50% of the crimes were perpetrated upon children below the age of 14. For the most part, this did not involve sexual intercourse. About one-half were returned to the courts after a 90-day observation period as untreatable. Of the remainder, the results of an intensive treatment program proved gratifying—centring mainly on group psychotherapy. Three-quarters of the group were discharged as improved and preliminary studies showed a low percentage of recidivism.

In contrast, Glueck[8] studied 30 pedophiles, 30 rapists, and 50 non-offenders as controls. Among other features, a serious impairment of social and moral conscience formation and resultant impairment of the restraining effect of conscience on overt behaviour was noted (this smacks strongly of the psychopathic personality). Because of the *lack* of improvement in the majority of men treated in individual group therapy in his series, organic therapies of various types were employed. The immediate post-treatment results in a group of sex offenders treated intensively with electroconvulsive

therapy showed more promise than other measures. This is remarkable in view of the fact that few psychiatrists would ever consider this treatment for sexual deviation *per se*. The author noted that the serious character of the psychopathology in sexual offenders made psychotherapeutic approaches quite ineffective. In psychiatric research, particularly in the therapeutic area, personal bias and faith by the worker involved, often plays a part in the outcome, so far as opinion about "improvement" is concerned.

The Forensic Clinic in Toronto reported a high percentage of failed appointments in 1958[2]—23% as against actual attendance. A similar finding is noted in the 1959 Annual Report from the clinic. This is not surprising in view of the high degree of character disorder present. Such persons often fail to develop close relationships with their doctors in a treatment program, and often fail to accept responsibilities of many kinds, including the keeping of appointments. The majority of new patients in 1958 were young; only 10% were over 40 years, 24% being between 20 and 24 years, and 20% between 16 and 19 years.

In group therapy with voluntarily referred homosexuals,[9] sexual relationships between members have developed. One aggressive member had to leave when he began acting out against passive members. Difficulties developed because of strong resistances offered by many men with character disorders. It was felt that a few, but by no means all of the group, were being definitely benefited by treatment.

There seemed to be general improvement in a group of exhibitionists, and also in a group of pedophiles, but repeat offences took place, although the incidence was low. Eight of twelve voyeurists were treated, and three improved.

The Clinic, created originally under the leadership of Dr. Peter Thomson, and now directed by his successor, Dr. Edward Turner, is enthusiastic about the future, in terms of its potential for research, teaching and treatment.[10]

A few typical case histories, involving the more common type of sex crime, are given below, taken from the author's personal files. It is apparent in most cases how prevalent the theme of masculine inferiority appears.

CASE 1.—A 27-year-old artist and office worker, a British Army veteran, was referred from the courts because of exhibitionism. He was reported to the police after exposure to a female guest he had never seen before, at a social gathering one evening. He was married with one child, intelligent, personable and well motivated. His domineering and hostile father always told the patient as a boy, "You'll never amount to anything! You'll never be much of a man." He was handsome, of athletic build, short, and always timid in locker rooms about exposing himself to other men. He had a history of periodic exhibitionism with erect penis to women of varied ages—without concern for intercourse, although it might be offered by the "victim". A shocked expression would be his greatest delight. He was, in effect, saying—"Look at me—I really am a man, am I not?" He experienced great concern about feelings of inadequacy. There was some improvement with psychotherapy, in that the patient ceased to exhibit himself, and he expressed improvement in his feelings of his own masculinity and personal confidence.

CASE 2.—A 44-year-old member of the Armed Forces was referred in lieu of a prison sentence after making improper advances and suggestions to a neighbour's 12-year-old daughter. Her parents were most generous in not pressing charges, provided he sought treatment. A "Don Juan" as a younger man, he had continually to assert his masculinity; by this it is meant that he was sexually promiscuous, having innumerable affairs with successive partners. Such men have profound but unconscious doubts about their abilities as lovers, and continually have to keep proving to themselves that they are adequate in this regard, by successive ventures (e.g. the career "playboy" type of individual). He had been drunk (and therefore with lowered inhibitions) at the time of the offence. His first wife divorced him because he committed adultery. His second wife was frigid and denied him intercourse because of dyspareunia and gynecological difficulties. A combination of "ageing" plus unavailability of partners plus sexual frustration led to the seeking of an "easy mark". Although he claimed amnesia for the event, witnesses stated he was not deeply inebriated, so this was likely malingering to save face. His marked depression and guilt afterwards, plus lack of assaultiveness in experience with women before, suggested a favourable prognosis. His reactive depression seemed genuine. In interviews, he revealed long-term feelings of personal inferiority comparing himself with other men.

CASE 3.—A 29-year-old clerk from Edmonton was referred because of homosexual invitations to other men. He had much anxiety over his homosexual tendencies. His usual contact consisted of his being masturbated by his partner, with no other activity involved. Outlets would always be sought whenever he was depressed over his financial and situational difficulties, at which times he would feel inferior, inadequate and not enough of a "man". He was married, with two children. He felt that he was feminine in voice and appearance and wished he might lose his hair because it was thick and curly, and possibly feminine-appearing to others. He was actually not at all effeminate-looking. He experienced marked prevalent feelings of inadequacy and inferiority. His greatest thrill in being masturbated was to have his penis *pulled* upon. In effect, he was unconsciously asking other men to castrate him and therefore feminize him, i.e. pull his penis away from his body. Early in his course of therapy, he kept asking for "surgical" operation to his genitals, i.e. castration.

The strong feelings of reactive anxiety seen initially in treatment suggested that the long-term prognosis was favourable. At the time of writing, this man had been under treatment for ten months. Whenever he did seek occasional homosexual outlets after therapy began, it always followed a period of frustration over financial difficulties, strained personal relationships at work, and so on, wherein he would feel most inade-

quate and unworthy. The frequency of such contacts was markedly reduced as a result of psychotherapy. As well, his feelings of personal worth and adequacy and feelings of masculine self-esteem improved considerably.

CASE 4.—A 15-year-old youth was referred from the Edmonton Juvenile Court Detention Home after it was reported to police that he had removed the underclothing from a four-year-old girl behind his house and apparently ejaculated upon her, frightening her to an extreme degree. This had happened at least once before with the same child, and the reddened appearance of her external genitals, as seen by a medical examiner, confirmed her story. The youth denied the charge, stating that he took her there to show her his rabbits, and that one of them had urinated upon her and one upon him at the same time. He was quite sullen, withdrawn and hostile and had a poor school record (several failed terms) with apparent retarded level of intelligence. His voice was high-pitched. His social behaviour and clinical record strongly suggested the picture of an incipient schizophrenic breakdown. Because of this, the gravity of the problem, and his denial of, and inability to appreciate the whole issue, as well as his intractability to the interview situation, it was strongly recommended that he be detained under observation in the Oliver Mental Institute as the immediate prognosis and potential response to treatment was unfavourable. Inability of his mother to grasp the severity of the situation also underlined the necessity of barring his return to the community, and emphasized the need for residential treatment. She saw him only as a "good boy", and planned to send him to university ultimately.

CASE 5.—An 18-year-old student barber admitted great anxiety over his attraction to other men in which he fantasied himself in, or behaved actually as the female partner, in intercrural intercourse. He was extremely boyish-looking, of low average intelligence and quite effeminate in appearance and behaviour. He exhibited very little spontaneity and was quite shy and introverted in the interview situation. This problem had been present since the onset of puberty. The heavy constitutional element in this case, plus poor material as a long-term psychotherapeutic case, all led to the erroneous estimate of a poor prognosis. He experienced strong feelings of personal inadequacy and inferiority both as an individual and in the social scene, and was most uncomfortable in the presence of girls. Previous individual and group therapy had failed. Psychotherapeutic interviews were directed at bolstering this lad's feelings about himself as a person, and in the course of treatment the therapist became a decided paternal figure to him. With the help of meprobamate medication and such psychological support, his symptoms of anxiety and depression abated, rumination about himself as feminine-like lessened in intensity, and his interest in girls became much stronger. As well, he began going out on dates with the opposite sex, and developed much more self-confidence in general.

CASE 6.—A 39-year-old merchant, several years before, had been charged in Juvenile Court with indecent assault concerning a minor. Many young children frequented his store and on occasion he would be attracted to preschool-age girls, taking their hands and placing them upon his genitals through his trousers. When one such child reported him to her parents, he was arrested, and subsequently referred for therapy. He was a respectable member of the community, was married and had two children. Always shy of women, he had entered marriage uninitiated to sexual activity. In the Armed Forces, he had much opportunity for intercourse but was never able to "follow through". On one occasion he slept all night with a prostitute, but could not "work up the nerve" to have relations with her. In treatment by two successive psychiatrists he lost his interest in female children and coincidentally developed an improved attitude in his feelings about his own masculine abilities. As well, his fantasies about sex moved from prostitutes to women of his own social status, and in fact lately to a possible affair with a sister-in-law. The latter was not attempted, but at least indicated a progressive "maturity" in sexual yearnings. Initially he had selected rather "helpless" partners who would offer no resistance. In sexual relations with his wife he had always been passive and had never taken the initiative.

CASE 7.—A 20-year-old service man, in the army 1½ years, was arrested because of indecent exposure to a young married woman in the laneway outside her yard. This occurred on impulse; he had never seen her before. Referred after being placed on probation, he admitted that for several years he had been stealing women's underclothing from clothes lines and that he had used these to excite himself during masturbation, ejaculating into them and then destroying them. He used to steal his own sisters' underclothing for this practice. He was one of four children, the others all girls. He admitted morbid shyness in the company of women. On the three occasions he had had intercourse, he always needed liquor to fortify himself. He admitted to a heightened feeling of sensual pleasure during the brief exposure, but felt "stupid" afterwards. His mother, rather than his father, used to administer beatings and punishments when he was younger. He stated that he used only *used* women's underclothing, never brand-new. This patient exhibited combined perversion in the forms of fetishism and exhibitionism. The ultimate prognosis was thought to be poor in view of the schizoid make-up of the patient, and his dull intellectual level. He was described by his superiors as being sheepish and an inadequate soldier. Several months after his original referral, in which time follow-up interviews were carried out, there had been no further attempt to carry out either act. Likely, fear of punishment was a strong deterrent factor in this case.

DISCUSSION

The number of children and adults who die annually from sex crimes is minimal, compared with the number who die from other crimes, accidents and diseases. There is a common misconception on the part of the public that exposure to sex deviates in childhood, of itself, will lead to later neurosis or emotional and/or sexual maladjustment. Isolated episodes such as these do not likely have lasting effects, if the child involved is basically emotionally healthy. Prevalent concern over sexual sensationalism is a characteristic of our society, and may well belie our own inner anxiety about sexual matters. To think of sex deviates as

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being "ill" and requiring treatment as well as, or rather than, a prison sentence is humane.

Since most sex deviates are *not* inclined to violence or assault (excluding rapists and deviates with sadistic tendencies), there would seem to be no need for mass removal of such persons from the community for a prolonged period of time. A great deal needs to be done as yet regarding public education in this matter, and it goes without saying that a wealth of research is yet required in this field. Research helps often to determine *what* is going on, but unfortunately does not always tell us what to do about it.

The problem of sex deviation and the sex criminal is not unlike other social and medical problems. Constitutional and environmental factors are likely both present in varying degrees in each offender. Cultural, religious, philosophical, moral and economic factors may well play a part in varying degrees. As an example of the latter factor, a case is described by a colleague, Major Carleton Taylor of Edmonton. This involved a serviceman who was befriended by a wealthy homosexual from whom he later received financial remuneration after granting this person homosexual favours. This was done because the serviceman, who had no serious homosexual interests, was badly in need of money because of numerous gifts he had purchased for his mistress at that time. He was apprehended and subsequently discharged from the service.

Johnson and Robinson[11] feel that perverse sexual behaviour develops from unconscious or, less frequently, conscious pathological fostering of deviant sexual behaviour, early in life, within the family setting. A parent may either act openly seductive towards a child, or may, by equivocation, foster behaviour oriented towards sexual deviation. This writer agrees with their opinion that definitive psychiatric therapy is a prodigious task in both the adolescent and adult sexual deviant, and that *it is futile in patients who show no compelling motivation for treatment.* They feel that there should be education of well-intentioned parents who do not observe proper and healthy rules of modesty in the home. They admonish pediatricians and family physicians to face the unequivocal truth that all degrees of seduction and sexual stimulation occur all too commonly in the home, and in families which exhibit every *outward* aspect of respectability, decency, and conformity with convention.

Ellis, Brancale and Doorbar[12] of the New Jersey Diagnostic Center for Sex Offenders suggest certain recommendations for the prevention of sex crimes: (1) To reduce drastically the number of sex offences on the statute book (e.g. why should sex perversion between two *mutually consenting* adults be considered a crime?). (2) To increase the amount and objectivity of sex instruction. (3) To encourage a much more liberal, socially sanctioned heterosexual participation on the part of young people.

(The author of this paper does not necessarily agree with the last recommendation of Ellis *et al.*, in terms of its potential to reduce the number of sex crimes.)

The core of sex crime, then, is that of inability to appreciate oneself as being "masculine" in one's own eyes, and to be inadequate sexually to a degree that one is unable to form satisfactory relationships with the opposite sex. There may be marked hostility directed to women in general, or the individual involved may derive sensual pleasure from inflicting violence upon others. Should this hostility erupt in an uncontrolled fashion, the beating of one's partners may take place, and rape, or the murdering of one's victim—a woman or child —may be the outcome. Such assaultive sex crimes comprise only a minority of all sex crimes, but these naturally receive the major share of publicity in the lay press.

Let it be stressed that all sex deviates are not sex criminals, e.g. a fetishist is not a sex "criminal" as such. Certain types of deviation are listed in the criminal code, and if a deviate commits one such type of act he becomes a sex criminal, whether or not he is apprehended. The majority of sex criminals are not dangerous in terms of being potential murderers, contrary to popular belief. In criminal sex acts, attention is usually "forced" upon the victim—as in the case of rape, exhibitionism and pedophilia.

When criminal sexual behaviour takes place, it is perpetrated upon a woman, a man or a child, but in all cases, sexual gratification is attained from a goal less than the ideal, mutually acceptable, heterosexual relationship. Hopefully, such a heterosexual relationship is complete with sincerity, mutual respect, compassion and love.

To hope that in the immediate future all sex deviates and sex criminals can be "cured" with "treatment" is at the present time to be naïve and idealistic. We are undoubtedly heading in the right direction, but society's hoped-for goal in this problem area remains as yet distant, and perhaps not completely attainable.

SUMMARY AND CONCLUSIONS

Sex criminals are usually men with underlying feelings of inadequacy regarding their own masculine status, and so seek less than an ideal heterosexual relationship with a mature woman as an optimum sexual outlet. This psychopathology is for the most part below the level of consciousness: the offender has incomplete awareness of his feelings and attitudes in this area.

The personality makeup of the majority of persons guilty of sex crimes, often, but not always, contains strong evidence of psychopathic behaviour, inferring relative lack of social and moral conscience. This augurs poorly for a successful treatment outcome in these particular men. Where the basic personality is not so affected, the prognosis is more favourable. It would be fair to say that the prognosis for the tendency to deviate sexually is the prognosis of the underlying personality or personality disorder.

Several case histories involving the most typical kinds of sex deviation—homosexuality, exhibitionism, pedophilia—are presented, and in these cases there is evidence of a prevailing "castration anxiety" with feelings of masculine inferiority.

The problem in general, involving causation, clinical findings, results of investigations and treatment, and social implications, is discussed. It is stressed that there is no one single objective form of treatment for sex deviates, criminal or otherwise. Results vary between different workers. Group and individual psychotherapies are the major forms of treatment available. Many of the strongly psychopathic personalities involved in sex crime are unfortunately refractory to such measures.

The author wishes to express his gratitude to Dr. Keith A. Yonge, Professor and Director of the Department of Psychiatry, University of Alberta, Faculty of Medicine, and the University of Alberta Hospital, for his helpful criticisms and recommendations regarding the writing of this paper, and to Mrs. Sheila Harrison and Miss Betty Davies for their assistance in preparing the manuscript.

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RÉSUMÉ

A la base du crime sexuel est l'incapabilité de l'individu de s'accepter comme être entièrement masculin et une insuffisance sexuelle assez prononcée à rendre impossible des relations satisfaisantes avec l'autre sexe. On trouve ou une hostilité prononcée contre les femmes en général ou l'individu en question prend un plaisir sensuel de l'emploi de violence contre des autres. Si cette hostilité éclate sans restraints, il est possible que le partenaire soit battu et un viol ou le meurtre du victime—une femme ou un enfant—peut en résulter. Les crimes sexuels violents de ce genre ne forme qu'une minorité parmi tous les crimes sexuels, mais naturellement ils reçoivent la plus grande publicité dans la presse laïque.

Un conduite sexuelle anomale est donc infligée à une femme, à un homme ou à un enfant, mais dans tous les cas la satisfaction sexuelle est obtenue par un but inférieur à la relation hétérosexuelle idéale et mutuellement acceptable. Dans les meilleurs cas les relations hétérosexuelles sont pleines de sincérité, de respect mutuel et d'un amour tendre.

En général cette psychopathologie se trouve au dessous du niveau de conscience et par conséquent la malade ne se rend compte qu'insuffisament de ses attitudes et sentiments sexuels. Si le malade souffre d'une personalité évidemment pathologique, c'est-à-dire s'il a des défauts du sens social et moral, les résultats de traitement sont souvent très médiocre. La psychothérapie individuelle ou en groupes et la méthode principale de traitement qui se trouve à notre disposition. Un seul mode de traitement objectif des perversions sexuelles, qu'elles soient criminelles ou non, n'existe pas. Les résultats de thérapie diffèrent d'auteur à auteur.

Le problème du crime sexuel en général est discuté, y compris les causes, les signes cliniques, les résultats des investigations et traitements et les conséquences sociales. Quelques dossiers des cas les plus typique de perversion sexuelle, à savoir l'homosexualité, l'exhibitionisme et la pédophilie, sont présentés.

Il est souligne que toute thérapie psychiatrique définitive d'un pervers sexuel, adolescent ou adulte, est une tâche énorme et qu'elle est en vain chez des malades sans désir sincère de se faire traiter.

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PAGES OUT OF THE PAST: FROM THE JOURNAL OF FIFTY YEARS AGO

THE MARITIME MEDICAL NEWS

In the *Maritime Medical News* for December an important announcement is made to its readers, which with all proper humility and thankfulness we reproduce in part.

"There has been, for a long time, a feeling that we in Canada should have a journal, somewhat on the lines of the *British Medical Journal*, a journal which should serve the interests of the profession throughout the Dominion. It was natural that the impulse should come first from the Canadian Medical Association. Whether or not, in course of time, the Association may, as in the case of the British Medical Association, be represented in every part of the country by branches, forming the local medical societies, there can be no doubt that a journal conducted by the Canadian Medical Association, publishing the papers read at its annual meetings, and such other articles as might be contributed, discussing matters of importance to the whole profession, such as medical reciprocity, giving from time to time a conspectus of medical progress in general, and such items of home and foreign medical news as would be interesting to us all, would be at once an interesting paper and a powerful factor in binding our scattered interests in one Canadian whole. More than a year ago, at the Winnipeg meeting, sanguine spirits hoped for the appearance of such a journal during the present year. There were lions in the way and it is only after an immense amount of hard work and a great deal of mutual concession that the Finance Committee of the Canadian Medical Association is in a position to issue the circular we have just quoted. Knowing, as we do, the high aims of those who have struggled so hard to found this journal, and the distinguished ability of those directly in charge of it, we have great confidence in urging all our readers to subscribe for it. And this brings us to consider our own relations to the new journal.

"The *Maritime Medical News* was founded in 1888 by Dr. Arthur Morrow, now residing in Kalispell, Montana, and has, we believe, served a useful purpose, and has been, to some degree, a bond of union to the profession in the Maritime Provinces. It must be evident that the new journal, if successful in its aims, will serve a much wider purpose and must tend to unify the interests of our profession throughout the Dominion. A careful consideration of all these circumstances has led the shareholders and editors of the *Maritime Medical News* to the conclusion that it is their duty to further, as far as possible, the interests of the new journal, as they believe their interests are also those of the medical men of Canada, and they have, therefore, resolved to suspend the publication of the *Maritime Medical News* with the current issue. If, at some future time, it should appear to be in the interests of the profession in these Maritime Provinces to have a journal representing local and special needs, we have no doubt men will be found able and willing to resuscitate and revivify this slender body of ours. And now to all our readers a kindly farewell! To the new Journal our hearty and loyal good wishes, and to all our comrades from Sydney to Victoria (and not forgetting Newfoundland) a Happy New Year!"—Editorial, *Canadian Medical Association Journal*, 1: 58, January 1911.

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USE OF PHENELZINE (NARDIL) IN A GENERAL HOSPITAL SETTING*

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UNTIL RECENTLY, psychic depression has been treated with psychotherapy, electroconvulsive therapy and time. In the past several years, groups of therapeutic compounds have been introduced which appear to have a specific euphoriant effect upon depressed patients.

One such group, the monoamine oxidase inhibitors, includes iproniazid (Marsilid) and phenelzine (Nardil). Phenelzine is B-phenylethyl-hydrazine dihydrogen sulfate and is related chemically to nicotinic acid, niacinamide and iproniazid.[2] It is dispensed in 15-mg. tablets with a recommended dosage range from 30 to 75 mg. daily. Recent reports[2-8] indicate that it is of value in the management of depressive illnesses, with an advantage that side effects are few and mild.

Monoamine oxidase is an enzyme required for the metabolic destruction of serotonin.[1, 2, 7] Inhibition of monoamine oxidase by iproniazid or phenelzine in animals causes a rise of cerebral serotonin concentration.[2, 7] It is assumed that monoamine oxidase inhibitors cause a rise in serotonin in humans, although no definite relationship is known between this substance and mental depression. Similarly, the reason for mood elevation with monoamine oxidase inhibitors is still obscure.

Dickel *et al.*[5] reported 80% favourable response in 85 outpatients. Arnow[7] reported 66% remission in endogenous depression and affective psychosis, with lesser responses in neuroses and schizophrenia. Saunders[3] reported best results in pure depressions but some response also in schizophrenia. Thal[6] reported that 80% of patients with depressions recovered within 60 to 90 days, and Sarwer-Foner *et al.*[4] successfully treated 42 of 60 patients suffering from neurotic depressions. Furst[2] reported 50% remission in 50 non-hospitalized depressed patients.

CASE MATERIAL AND METHOD

A total of 68 patients, 28 male and 40 female, with acute and chronic illnesses, were tested. Many of these patients had received previous long-term psychotherapeutic and physical assistance. Their diagnostic categories were as follows: manic-depressive, 6 patients; involutional reaction, 12; reactive depression, 21; schizophrenia, 15; and mixed psychoneuroses, 14.

Treatment began with two or three phenelzine tablets daily, increased to five or six tablets if insufficient response occurred. If anxiety or agitation was present, a tranquillizer was administered as well (26 patients). A trial of at least two weeks was considered necessary before discontinuing the drug, unless severe side effects occurred. In spite of reports[2-8] that phenelzine has no specific hepatic or hemopoietic toxicity, spot checks of these functions were performed on hospitalized patients. Electroconvulsive therapy (E.C.T.) was used concurrently on three patients, these being considered only for determination of side effects, not for therapeutic assessment of the drug.

RESULTS

Sixteen patients (25%) attained complete recovery, and in 21 patients (32%) improvement was sufficient to permit discharge or effective performance with residual symptoms. Noticeable improvement was recorded in 8 patients (12%), and in 20 patients (31%) there was no response. Thus, 57% of the 65 patients recovered sufficiently to be discharged from hospital or regained effective performance as outpatients.

The remission rate considered according to diagnostic category was: manic-depressive (depressed), 100%; involution melancholia, 75%; reactive depression, 48%; schizophrenia, 33%; mixed psychoneuroses, 60%. In the four cases in which electroconvulsive therapy had failed previously, no success was gained with phenelzine. On the other hand, of the seven phenelzine failures, five were able to be discharged after ECT.

Side Effects

Side effects were frequent but were usually not of sufficient severity to interfere seriously with the progress of treatment. They were present in 18 cases: lightheadedness and dizziness, in 7 patients; hypomania, 5; ankle edema, 3; mild urinary retention, 2; fatigue, 2; irritable outbursts, 2; hypotension and fainting, 2; impotence, 1; bad taste in mouth, 2. Treatment was discontinued in two patients because of hypotension with fainting.

No cases of jaundice, liver impairment or blood dyscrasia were noted, either clinically or in laboratory studies.

Time Before Improvement

Frequently, improvement was striking during the first week of administration of phenelzine, but this was more common during the second week. In some cases, recovery was sudden; in others, it was gradual. In five cases, initial improvement was temporary followed by a partial relapse, proceeding to a successful outcome.

DISCUSSION

The results in this series indicate an excellent response in the depressed phase of the manic-depressive illness. Three of the hypomanic reactions were in manic-depressive patients; patients with this reaction were easily controlled by tranquil-

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lizers. Adjustment of the dosage or discontinuing the medication was sufficient.

Adequate response was obtained in involutional depressions. Phenelzine appeared of lesser value in reactive depressions, schizophrenia and mixed psychoneuroses but had considerable effect on some individual patients.

Toxic reactions were not a serious handicap, the most troublesome being unsteadiness and dizziness, usually not necessitating withdrawal of the drug. Urinary retention developed in one patient during treatment, but further investigation revealed a previous history of this symptom under stress. A homosexual trend was found in the one patient who complained of heterosexual impotence.

Use of tranquillizers is considered necessary where anxiety or agitation is prominent or where suicidal threat is present. The latter may be an indication for electroconvulsive therapy in severely depressed cases.

SUMMARY AND CONCLUSIONS

Phenelzine (Nardil) produced remission in 57% of 65 depressed patients in a general hospital psychiatric practice. It appeared to be beneficial in the purer depressions, such as manic-depressive illness (100%) and involutional melancholia (75%). Reactive depressions and depressions in patients with schizophrenia and psychoneurosis responded less frequently, but often gratifying remission was obtained. Toxic reactions, although common, are not a major deterrent to administration of phenelzine. Simultaneous administration of tranquillizers may be of value.

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MEDICO-LEGAL

MEDICAL MALPRACTICE LITIGATION—THE DOCTORS' DILEMMA*

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PART III OF FIVE PARTS

MALPRACTICE LITIGATION AS A CURRENT PROBLEM IN MEDICINE

The Magnitude of the Problem

IT HAS BEEN stated at the outset (see Part I) that the incidence of malpractice litigation has in recent years been increasing at a rate which has justifiably alarmed the medical profession and presents it with a very definite problem. No less alarming has been the amount of the damages awarded in some cases. Although this problem has manifested itself most severely in the United States and to a lesser degree in England, it has also been felt in Canada and its very existence carries an inherent warning that merits its further study.

The magnitude of the problem may be judged by examining several factors. First, the number of malpractice actions initiated is a measure of the scope of this problem and has been sufficiently great to attract the attention of the popular press. A national news magazine reported a few years ago as follows:

> "Some 5000 cases are now being tried each year with thousands of other claims settled out of court. Since 1950, one out of every 35 doctors insured under the New York State Medical Society's group-insurance plan has been sued in the courts for malpractice."[43]

More recently another publication of national circulation emphasized the extent of the malpractice problem with this statement:

> "Currently it is estimated that similar malpractice claims are being filed at the rate of at least 6000 and possibly as many as 9000 a year. According to the American Medical Association one out of every seven doctors in the United States has been sued. In New York and the District of Columbia the ratio is one out of five. In California it is one out of every four."[44]

In an attempt to evaluate the extent of the malpractice litigation problem, Sandor[7] made a survey of all the appeal cases decided in the higher courts of the United States from 1794 to 1955 on issues of medical malpractice. This study was limited ex-

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pressly to appeals following civil action against medical doctors arising out of medical practice.

> "It should be borne in mind that the cases decided by the appellate courts represent only a small fraction of the actual number of cases filed in the lower courts. It would appear that for every 100 actions filed only one will eventually reach the appeal court."[7]

Since it was found that in the period under study 1936 appeal cases were decided, it follows that in all some 1,936,000 actions against doctors were launched. This in itself does not give a dynamic view of the magnitude of the problem. However, Sandor's study also illustrates by graph the incidence of civil malpractice appeals in the United States for the years 1900-1955. Although this incidence fluctuates, the upward trend has been persistent and, after a drop during the war years, the trend is again in the direction of more litigation.

If the rate at which doctors are being dragged into court offers alarm, no less discomfiting are the huge sums in damages being awarded to plaintiffs by American juries. In six recent malpractice cases heard in United States courts, verdicts were given in amounts ranging from $97,000 to $250,000. This too serves to illustrate the extent of the malpractice problem, for not only is there an increasing tendency for patients to sue doctors but there is also an increasing tendency for juries to award larger and larger sums in their verdicts.[7]

Another index of the seriousness of the problem is found in the changes which have occurred in malpractice insurance. Thirty years ago doctors carried only nominal protection against malpractice liability because plaintiffs were not often successful, and when successful, judgments were small. Today the physician often is covered to the extent of $100,000, or even more in the case of certain specialists. The premiums on these policies have been increasing over the past ten years and now may cost a doctor $400 to $600 per year.[44] Furthermore, some insurance companies in the United States have claimed that their losses from malpractice insurance have been too great and they have therefore stopped selling protection against this risk. It has been suggested that if this trend continues the question facing a doctor will not be what price he will pay for malpractice insurance but whether he can obtain it at all.

From the foregoing one can glimpse the dimensions of the present problem of medical malpractice litigation. In fact, these dimensions are so rapidly enlarging that it has been queried whether the law itself is changing.

> "The increase in the number of actions against hospitals and doctors has given rise to the feeling in some quarters that the law of negligence in so far as it affects doctors has undergone a drastic change."[2]

THE EFFECTS OF PROBLEMS

The effects of this rising tide of malpractice suits have manifested themselves in different ways. Firstly, the economic impact of the situation suggests itself readily. The large sums awarded in damages have already been mentioned. It has been reliably estimated that the costs of malpractice litigation to doctors and hospitals alone, in court judgments, out-of-court settlements, and legal fees, amount to at least $45,000,000 per year. The result has been an increase in the cost of malpractice insurance, an expense which has eventually been passed on to the patient.[44]

But the effect measured in dollars and cents shrinks in importance when one considers the effect on the practice of medicine. The ever-present threat of patients resorting to law has served to envelop medical practice in an atmosphere of fear—fear of being sued. Consequently, influenced by this apprehension, doctors tend to order additional diagnostic tests and to request medical consultation without clear indication. Furthermore, it has been suggested that doctors have become reluctant to perform certain valuable procedures in diagnosis and surgery because there is some risk attached. Dr. James W. Watts, Chairman of the Neurological Department of George Washington University Medical School, recently asked 20 board-certified specialists whether the threat of malpractice had made them wary of any particular procedures. Some of his findings were as follows:[45] One general surgeon replied he had abandoned the Wertheim radical hysterectomy for carcinoma because of risk of ureteral fistula. Two neurosurgeons stated that they no longer cut the vestibular nerve for Ménière's disease because of the risk of facial nerve injury. Three anesthesiologists reported that spinal anesthesia was being given less often because courts have ruled that the doctrine of *res ipsa loquitur* applies to neurological complications. This was admittedly a small sampling, and a survey conducted on a national scale confirms the suggestion that the threat of suit has led to diminished use of certain procedures only in the instance of spinal anesthesia.[44] However, that medical practice should be influenced from outside the profession by a threat cannot be anything but detrimental to both doctors and patients and poses a question of deep import. While it may be argued that more procedures and more consultation may benefit the patients through better diagnosis, such argument is spurious because the patient must bear the additional expense. Again, while it may be argued that the fear of suit will benefit medicine by prompting study and re-evaluation of some medical procedures to which risk attaches, it must be remembered that almost every medical procedure carries some risk, and in the procedures in question the value of the procedure is usually great and the risk small or the procedure would not be used in

any event. Therefore, any benefits derived by forced curtailment of certain procedures will be far outweighed by the resultant retardation in medical progress. Whatever the arguments may be for or against is immaterial in the face of the single shocking prospect, namely, that doctors apprehensive of the possibility of malpractice suits against them may act in the course of their practice, positively or negatively, not out of medical considerations related to their patient's welfare but out of legal considerations related to their own protection. That the doctors' apprehension of suit is a real one is seen in the fact that the vast majority of malpractice actions are brought in the absence of any negligence on the part of the defendant doctor.

> "Only about one in seven or eight of the district cases [District of Columbia] shows any voluntary negligence on the doctor's part. Nonetheless, the doctor loses about one in four cases."[2]

Sadusk states that 90% of doctors who are sued are not guilty of technical error.[4] The net result of this invidious situation in which doctors practise medicine with one eye on the court room is not the practice of the best medicine but rather the practice of timorous, overly cautious, overly circumspect medicine with less benefit and greater cost to the patients. As stated recently by an English Court:

> "We should be doing a disservice to the community if we imposed liability on hospitals and doctors for everything that happens to go wrong. Doctors would be led to think more of their own safety than the good of their patients, initiative would be stifled and confidence shaken."[46]

Another unfortunate facet of the malpractice problem has been the rise of antagonism between the medical and legal professions.[47] This disharmony between two great professions is particularly distressing in view of the fact that a great percentage of litigation today involves claims of personal injury in the disposition of which both physicians and lawyers must function to achieve just solutions. What are the grievances of the one profession against the other? The main source of friction is the doctors' belief that the lawyers are hostile to them and are largely responsible for the mounting wave of malpractice suits and the huge verdicts awarded in them. On the other hand the lawyers complain that the doctors refuse to testify as expert witnesses in malpractice cases even where malpractice is obvious.[48] To what extent are these grievances justified? While the doctors' vision of the lawyers as a voracious, hostile horde may be exaggerated, there is in the United States an organized segment of the legal profession which specializes in representing plaintiffs in injury liability cases and pursues the objective of securing higher and higher jury awards with great zeal and apparent success.[49] This group is the 5000-member National Association of Claimants' Compensation Attorneys, which has drawn the fire of both the medical profession and defence attorneys. The N.A.C.C.A. has been accused of questionable courtroom tactics in which the plaintiffs' lawyers play on the emotions of the juries by the histrionic and gruesomely visual presentation of evidence. The result is to obscure the true issues of liability and damages, and both are determined not by the jury's reasonable evaluation of evidence but rather by the degree of its outrage at the severity of the injury. The N.A.C.C.A. offers no apologies for its tactics—in fact it conducts seminars on how to present evidence effectively—and it heralds the success of these tactics as long overdue. In the words of Mr. Melvin M. Belli of San Francisco, perhaps the most able, most successful, and in some quarters most feared malpractice lawyer:

> "I see those large judgments as an indication that justice is at long last being done – that a somewhere-near-adequate price tag is belatedly being placed on human suffering caused by human error."[50]

The legal profession is not free of criticism of its role in the malpractice problem from within its own ranks, and an eminent American professor of law points a finger of censure in these words:

> "The vast majority of medical malpractice cases would evaporate if lawyers would view them critically rather than hopefully. A few telephone calls, seeking objective truth rather than helpful evidence, would dispose of many such claims. To view every claim as *prima facie* valid (a 'case' with probability of settlement of any such 'case') is a perversion of the lawyer's function. He is an officer of the court, as well as an advocate for his client – to repeat the well-known phrase that so often receives lip service. What I am saying is nothing less than this: It is the duty of the lawyer to discourage malpractice suits generally, except where they are reasonably well founded, not merely out of courtesy to physicians, but as a matter of duty to the law, the courts, the legal profession, and to society."[47]

Before the medical profession condemns the legal profession or even a part of it out of hand and smugly concludes that here lies the problem and the answer to it, certain facts must be considered. The lawyers who act for patients in malpractice suits are fulfilling their roles as lawyers within the structure of their system of jurisprudence according to its laws and procedures. They are, in other words, performing their duties. It may well be that in the United States the

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frequency of jury trials on technical issues predisposes to verdicts for undue amounts of damages. Similarly, the legality of legal fees contingent on judgment in the United States may contribute to the frequency of suit and this may only be natural on considerations of human nature. In Canada, where issues of a sufficiently technical nature are more often removed from juries and decided by a judge alone, and where contingent legal fees are outlawed, it is submitted that the likelihood of suit and unreasonable judgments is lessened, but even so, by no means obviated. In any event, the medical profession as a whole would be wise to be wary of blaming the malpractice problem on the lawyers and to look elsewhere for etiological factors, perhaps at itself. A warning to this effect has been sounded by the Chief Executive Officer of the California Medical Association, an authority on malpractice:

> "Physicians at times are prone to believe that if only it were possible to muzzle the legal profession all malpractice suits would end. This approach overlooks the fact that an attorney cannot threaten or commence suit unless he has a client who is ready, willing, and able to sue. Overtures to the legal profession that in effect ask that profession to refrain from practising law within a particular field necessarily create resentment. It is the duty of a lawyer when retained to utilize all lawful means to advance his client's cause; if he does less than that he is not practising sound ethical law. He may even be guilty of legal malpractice."[6]

The lawyers have consistently lamented the difficulty of obtaining the services of doctors as expert witnesses. They have particularly bewailed the reluctance of doctors to act as witnesses for plaintiffs in malpractice suits, even where the malpractice is obvious. This reluctance they have labelled a "conspiracy of silence" whereby doctors, supposedly pressured by medical societies and insurance companies, seek to protect their negligent brethren.[51] It is unnecessary to raise the spectre of a conspiracy to point out that doctors are probably not, as a rule, willing witnesses, nor should one conclude that they are never willing. The doctors' attitude in this respect is quite natural.[47, 52] Can a doctor reasonably be expected to take pleasure in attacking in court a member of his own profession? Clearly he cannot do this without in the process undermining to some degree his own professional status. Even in giving testimony in a personal injury case a doctor who is witness for one party must necessarily impugn the knowledge and experience of the doctor witness for the other party. How much more distressing to the doctor is the prospect of directly impugning the professional ability of his fellow doctor who is defendant in a malpractice suit. Undoubtedly the greatest factor in the doctor's reluctance to testify is the fear of cross-examination. In his hospital the doctor is lord and master, accustomed to unswerving obedience. It is little wonder that he should wish to avoid exposure, in the lawyer's natural habitat of the courtroom, to the searching, probing, and often humiliating inquisition of clever counsel whose sole object is to cast doubt on his experience, knowledge, and opinions. Therefore, if the doctors drag their feet on the way to the witness stand, their inertia can be explained by their distaste for the task and ordeal they face there, and this distaste arises as an individual matter independent of "conspiracies".

However, merely understanding the doctor's point of view will not answer the problem of securing expert medical witnesses and it is clear that such witnesses are necessary. Fortunately, suggested solutions to this problem have been put forward, as will appear presently.

THE INVESTIGATION OF THE PROBLEM

The logical prerequisite of the solution to a given problem is a knowledge of its cause, and this holds true as much for the solution to the malpractice problem as it does for the treatment of a boil. If the frightening frequency of malpractice litigation induced alarm and bewilderment within the medical profession, it also spurred the profession to serious thoughts upon the causes of the problem. Sober reflection soon revealed the necessity for research and investigation.

Fittingly, this investigation began in California, which had the doubtful distinction of leading all the states in the number of suits filed per doctor, and the size of jury awards and out-of-court settlements. An important study was launched by the Alameda-Contra Costa County Medical Association in the San Francisco area. The report of this study, published in 1955, was based on a careful analysis of some 600 malpractice claims and suits filed in the two counties of Alameda and Contra Costa during the years 1946-1954.[53] These investigations were then extended under the direction of the California Medical Association to other counties in California, and a report of this statewide study based on 700 suits and claims was published in 1957.[54] Because the period covered in this statewide study was less than two years, its value is limited to the extent that it indicates trends throughout the state and to the extent that it confirms the earlier county study. Further research was carried out at a national level by the American Medical Association, which conducted an opinion survey on medical malpractice among approximately 7500 physicians, representing 5% of its membership.[55] Of this sampling a further questionnaire was sent to those physicians who indicated that a claim had been brought against them in the past. In addition the A.M.A. study included an analysis of all malpractice suits in the United

States which reached the Court of Appeals in the last 20 years, amounting to approximately 1000 cases. Finally, an opinion survey was conducted of all state and larger county medical societies to determine their experience with malpractice. The results of these studies are of interest not only because they unearth some concrete facts about malpractice liability but also because they are strikingly similar. Taking these investigations together, their findings disclose the following facts:[56]

1. There is no basic difference in the incidence of malpractice hazard between the general practitioner and the specialist.

2. The age and sex of the physician do not appear to be factors in the incidence of malpractice claims or suits.

3. The medical malpractice hazard depends not upon whether the physician is a general practitioner or specialist but rather upon the type of professional work done by the physician, i.e. upon the type of medical problem involved; approximately 60% of all malpractice claims and suits arise from some form of surgery.

4. The greatest number of surgical cases leading to malpractice claims and suits is in the field of obstetrics and gynecology, with orthopedics second and general surgery third.

5. The claimant or plaintiff is a woman in approximately 60% of cases.

6. The alleged negligent act in a malpractice claim or suit occurs in a hospital in approximately 70% of cases.

7. The hazard of malpractice litigation varies widely from hospital to hospital.

An important and interesting group of findings in these investigations concerned the reasons, primary and secondary, for which malpractice claims and suits were initiated. These reasons and the percentage of claims and suits in which they were the initiating factor are summarized as follows:

Primary Reasons	
Poor operative results	24.7%
Poor medical result	19.3%
Error in diagnosis	10.1%
Foreign bodies left in patient	8.6%
Burns	7.9%
Assault, lack of consent, abandonment	6.8%
Miscellaneous	22.6%
Secondary Reasons	
Careless comment by another physician	25.2%
Action prompted by attorney	15.4%
Claim brought for financial profit	13.8%
Patient had mental disability	7.9%
Action prompted by relatives	7.2%
Patient was an alcohol or narcotic addict	2.9%
Patient had another disease or disability which progressed badly	2.1%
Miscellaneous, not possible to classify	25.5%

Of these secondary causes which prompted the patient to claim or sue, it is seen that 27% are under the direct control of the medical profession and that 25% are related to the patients themselves.

A further interesting body of information revealed by these studies concerned the outcome of malpractice claims and suits. Of all malpractice claims which are initiated, 17% are dropped, 33% are settled or compromised, and 43% go on to become law suits. Of this 43%, 12% are dropped, 20% compromised, 9% decided in favour of plaintiff and 41% decided against claimant. It appears from this that while a substantial number of both claims and suits are dropped, of the cases which come to court a large number are decided in favour of the defendant doctor. At the same time a propensity to settle claims and suits is evident in the fact that 33% of all claims and 29% of all suits terminate with some money payment to the claimant or plaintiff.

The Alameda-Contra Costa study had made the startling disclosure that there existed in the physician population a hard core of doctors who might be described as "suit-prone". This description derived from the fact that this small group comprising about 1% of the physicians seemed to be involved repeatedly in malpractice claims and suits accounting for as much as 27% of damages demanded. The surprising discovery that such a core of "suit-prone" doctors existed was confirmed by the statewide study. This fact, considered in the light of the secondary causes of malpractice suits, directed the investigations on a new and promising tack, namely research into the psychological background of malpractice litigation. The need for such research has been well expressed by the former Chairman of the Medical Review and Advisory Board of the California Medical Association:

> "While actual malpractice must be prevented, it is not the major cause of claims and suits. Since 90% of doctors who are sued are not guilty of technical error, and since many doctors who are guilty of known error are often not sued by their patients, we must look at the doctor-patient relationship for the major causes of claims and suits. The growing malpractice problem is primarily a human relations problem and requires human relations research."[4]

This "human relations research" took the form of a series of studies of the psychological and circumstantial background of malpractice suits conducted for the Medical Review and Advisory Board of the California Medical Association. These studies were directed by a psychologist, Dr. Richard H. Blum, whose revealing report, "The Psychology of Malpractice Suits", was issued in 1957.[4] The conclusion drawn by Dr. Blum after more than two years of interviews and tests on some 2000 patients and 500 doctors was that malpractice suits represent a breakdown in the doctor-patient relationship and this may be stated as the basic cause of the so-called malpractice problem. The Blum report dealt at length not only with the

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suit-prone physicians but also with suit-prone patients.

These two groups were defined, for purposes of the study, as patients who had sued and physicians who had been sued more than once and were investigated for basic psychological characteristics which set them apart from patients and physicians who were not suit-prone. What are these basic psychological differences? The suit-prone patient, it was found, is characterized by attitudes which reflect a basic immaturity that is revealed in all aspects of the patient's life. Like a wilful child, he is always ready to blame somebody else for everything that goes wrong in his own life, and the doctor who is sued by such a patient is only one in a long line of victims of such projection. The suit-prone patient, it appears, has unconscious fears of illness, doctors, and death which manifest themselves in unreasonable and immature attitudes. Thus, he believes that he should recover quickly from his illness but is outraged by the necessity of paying the doctor; he mistrusts doctors but retains a childlike faith in the efficacy of medical science. It is readily seen that the immature emotional attitudes and unconscious fears of the suit-prone patient form a fertile soil for the seeds of anger and dissatisfaction, and the unfortunate harvest is often a malpractice suit. Because he expects too much of his doctor and is ready to blame him when something goes wrong, the suit-prone patient can readily find causes of dissatisfaction with which to reproach his doctor; for example, chronic illness, inability to establish a final diagnosis, ineffective predications, failure of surgery to bring about a complete cure, or unlooked-for side effects of treatment. "Once the patient thinks that the doctor has failed him, neglected him or hurt him, once the patient sees that his expectations are unfulfilled, that his fear and sadness continue, then it is easy for the patient to decide that malpractice has occurred."[4] It is clear that a conclusion by such a patient that malpractice has occurred will be arrived at more readily in the presence of some dramatic incident such as a surgical accident or an obviously unsatisfactory result. But even in the absence of such incidents the reaction of the suit-prone patient is conditioned by his immature psychological structure, and his decision to sue is based upon his own interpretation of his experience with the doctor.

But what of the suit-prone physician? What features set him apart psychologically from his fellow physicians who are not suit-prone? The suit-prone physician appears to be characterized by insecurity and immaturity. He uses his role as a doctor as a defence mechanism to shore up his faltering ego and to this end he needs his patients' admiration and gratitude. In the words of the Blum Report his psychological structure is summarized as follows:

> "The suit-prone doctor who is himself immature encourages patient immaturity and dependency. He wants patients to look up to him and put their hopes in him. The doctor tries to promise the patients what they want; he fears to admit to them or to himself his own inabilities or inadequacy. He does not warn patients of things that can go wrong nor does he explain the problems in medical care to the patient. He tries to do things which he is not equipped to do, and he finds it hard to turn to his colleagues for the assistance they could and should provide in the care of his patients.
>
> "The suit-prone physician has little understanding or awareness of his personality problems. He does not see how he distorts the doctor-patient relationship to satisfy his own unconscious needs. In many respects he is rather like the suit-prone patient. He does not take responsibility for the things that go wrong, he blames others for troubles and in a self-centred manner he gives priority to the feeding of his own needs before those of his patients."[4]

Faced with a suit-prone patient even the most competent and thoughtful physician may find himself enmeshed in the coils of a malpractice suit. But when a suit-prone patient and a suit-prone physician come together the situation is one of potential explosion which any spark of dissatisfaction may ignite, and chances of malpractice litigation ensuing in such case are found to be multiplied many times. The report suggests that the doctor may all too often add fuel to the fire by his failure to treat the emotions of the patient at the moment of dissatisfaction. Rather than sympathize with the patient and attempt to discuss constructively the patient's complaint, he may aggravate the patient's attitude by ignoring or rejecting him while continuing to send his bill. Let it be emphasized that no real malpractice need have occurred as the basis of malpractice suits involving suit-prone patients and suit-prone physicians. While there is reason to believe that suit-prone doctors do make more mistakes *per se* and that they are the ones who tend to be sued by suit-prone patients, most of the suits filed are not warranted complaints based on actual errors but on errors as interpreted by the patients.

The Blum report brings to light facts which strongly suggest the major cause of the increase in malpractice litigation. It permits the viewing of the problem against a backdrop of the psychological characteristics of the antagonists in malpractice suits and suggests how the clash of these characteristics produces situations which end in court. As stated by Dr. Blum:

"The malpractice story is a story of people. It shows how their personalities, their attitudes, and their ways of thinking and looking at the world are related to the legal actions that take place. It is also true that the two most important people in the malpractice suit are the patient and the doctor. Therefore, the malpractice story deals with these two people in particular. The story shows that malpractice suits are drastic symptoms of a breakdown in the relationship between the doctor and his patient."

The findings of these investigations that approximately 70% of all malpractice claims arose out of procedures carried out in hospitals is not in itself a startling disclosure when one considers that the serious and the difficult medical and surgical problems are usually dealt with in the hospital rather than in the office or in the patient's home. However, the fact as disclosed that more malpractice claims arose in some hospitals than in others merits consideration. For example, one hospital staff had seven times as many incidents per 100,000 admissions as that of another hospital staff in the same community. Why should this variability of incidence exist from institution to institution?

In an attempt to identify the characteristics of such "suit-prone" hospitals, the Medical Review and Advisory Board of the California Medical Association authorized a further study by Dr. Blum whose report, "Hospitals and Patient Dissatisfaction", was presented to the Board in 1958.[4] The findings in this report were based on investigations of five California hospitals who agreed to cooperate in the study.[57] Of these five hospitals two displayed a low incidence of malpractice suits against its staff and three a high incidence as revealed by the earlier investigations. The high-suit and the low-suit hospitals did not differ substantially in size, assets, or facilities and all are accredited by the Joint Commission on Accreditation of Hospitals of the American Hospitals Association, the American College of Physicians, the American College of Surgeons, and the American Medical Association. The low-suit hospital may be described generally as a small non-profit community hospital with a board of trustees who are well-educated community leaders; it has a malpractice-incident rate of 2.2 per 100,000 admissions. In contrast, the high-suit hospital is a district hospital (a semi-public institution built with state and local funds) whose trustees are elected officials, poorly educated, lacking in administrative experience, and subject to political influence; its malpractice incidence rate is 15.9 per 100,000 admissions.

However, the most significant findings in this report concern the relationships among doctors and laymen within these hospitals. These findings may be summarized as follows:

1. There are more personal friendships among doctors and trustees at the low-suit hospital than at the high-suit hospital.
2. Young doctors at the low-suit hospital do not believe they are "kept under" by their colleagues. But many of the staff men at the high-suit institution feel like perpetual underdogs.
3. There do not seem to be many personality clashes at the low-suit hospital, whereas they are a real problem at the high-suit hospital.
4. At the low-suit hospital, the executive committee runs things and influence is not important. Although the high-suit hospital is primarily run by its administrator, cliques and important personages sway the course of events.

There emerges from this report by Dr. Blum, as in his earlier one, a picture which relates the incidence of malpractice suits to the emotional and social atmosphere in which medical practice is carried on and to the interpersonal reactions of those who practise it. Whether a particular hospital setting is so poor that it produces a lower degree of conscientiousness among its doctors or whether the doctors are primarily the cause of a high-suit rate in a given hospital cannot be readily ascertained. In any case, there again becomes evident the existence of psychological factors among medical personnel which can be related to malpractice litigation.

The results of none of the enquiries mentioned are necessarily conclusive, although they were carried out by experts with meticulous care. Their importance lies in the way they illustrate the types of investigation which must be undertaken if the causes of the malpractice problem are to be clearly determined. The facts they disclose indicate potential curative measures which may be taken.

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CASE REPORTS

OLEOGRANULOMA: REPORT OF A BIZARRE CASE

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A GRANULOMA is an exaggerated physiological response within tissue provided by a foreign substance which withstands enzymatic destruction. To meet this definition a granuloma must contain unchanged or partly changed elements of a substance which has produced the tissue reaction.

In the body this normally manifests itself by an attempt at isolation of the substance with the construction of a connective tissue framework or cage about it. Such is the response to inorganic material lodged in the body and to organic products incapable of dissolution by tissue fluids. The extent to which this occurs is directly related to the *in situ* amount of foreign substance and the limiting anatomical factors.

The injection of paraffin to correct cosmetic defects is a sorry epic dating back to the turn of the century, and now lies buried in the trash heap of questionably inspired procedures. Paraffin C27H56 was the substance generally used. In the main its use was innocuous, but on occasion inflammatory reactions and tragi-comic disfigurements resulted. A few fatalities were directly attributed to the procedure, and it fell into general disrepute.

The unreliability of tissue reactions, and the frequent production of granulomas at the focal point of injection or remotely distant to it, were soon recognized by both pathologists and radiologists.

Generally, granulomas produced by paraffin, vegetable and mineral oils and resins are benign. These act as an irritant so that, once surrounded by living cells, connective tissue invades the liquid or semiliquid foreign material, converting it into a hard mass, with occasional calcification.

Oil granulomas of therapeutic origin have simulated cancer macroscopically, and have led to disastrously radical extirpative surgery.[2] Some of the liquid foreign material may be conveyed via the lymphatics to regional glands and form a mass similar to the masses encountered in metastatic lesions.

After cosmetic rhinoplasty, ointment-laden gauze packs inserted into the nares resulted in the formation of oleogranulomas necessitating secondary operation.[5]

Granulomas may develop soon after or years after the initial injection, and secondary spread may microscopically simulate tuberculosis. The typical appearance of oil vacuoles surrounded by the connecting stroma should prevent this error, but frequently a chronic inflammatory reaction with a periphery of lymphocytes and a sprinkling of giant cells is present and confusing.

"Apicolysis with plombage" was an early method of attempting an artificial collapse of a lung portion by inserting wax into the apex of the pleural cavity. The procedure fell into disfavour early and was supplanted by the simpler and more effective pneumothorax. Lung paraffinomas developed in a number of cases, and these often simulated primary or metastatic lesions radiographically. A paraffinoma of the mediastinum causing esophageal obstruction occurred six years after wax was used to produce an artificial pneumothorax.[6]

In the past, almond oil injections of hemorrhoids were common, and until very recently sodium morrhuate was used as a sclerosing agent in varicose veins, hydroceles, and varicoceles. Likewise penicillin and beeswax parenterally have produced granulomas. Grease monkey granulomas, also called oleomas, are of traumatic origin and in the same class. Oleopneumonia and oleoperitoneum are of this order and result from the lodgment of oils in tissues. The instillation of mineral oil directly into the peritoneal cavity for the purpose of preventing adhesions was a fairly common procedure. Mineral oil absorbed from the intestine may lodge in the mesenteric lymph nodes. Coppridge *et al.*[7] described a case of compression of the ureters by lipogranulomatous masses, resulting in bilateral hydronephrosis.

In some cases the content of the granuloma simulated modified fats and led to the description of an entity termed "sclerosing lipogranuloma" by Smetana and others. Newcomer *et al.*, after biochemical analysis of tissue, were convinced that these were all exogenous lipids.

In the case which is reported below, a seaman with curiosity and a flair for photography became the sorry victim of his own curiosity. Whilst on shore-leave in a West-African port, he attempted to photograph a tribal ritual. The natives considered his action sacrilegious, and his next recollection was that of finding himself in a hospital with his body mutilated and suppurating. His chest and abdominal walls, penis and scrotum had been perforated by means of sharp reeds and filled with vegetable oil. This was an obvious attempt to alter the sex characteristics superficially.

The event occurred in 1938, and 20 years later this man accidentally came to our notice. He had been too embarrassed to seek medical advice on his own initiative, but responded readily to the suggestion of plastic surgery. He has now undergone two stages of plastic repair, and only the abdominal panni remain to be corrected.

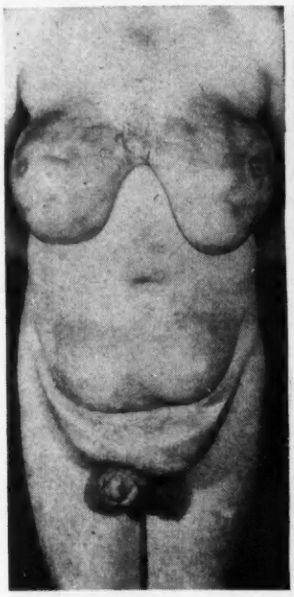


Fig. 1.

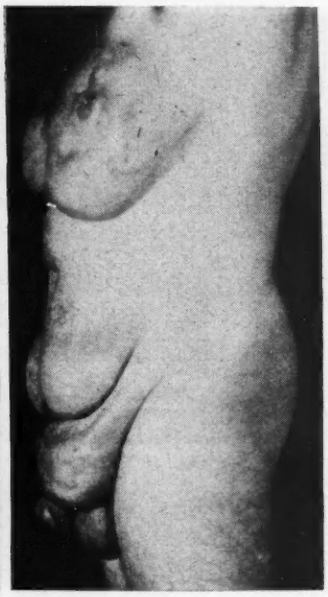
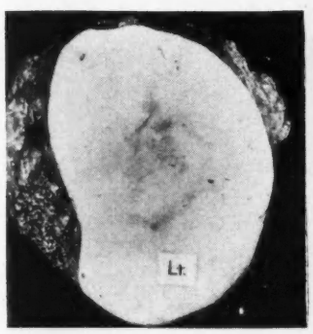


Fig. 2.

Figs. 1 and 2.—A.P. and lateral views showing pendulous scarred breasts, abdominal panni and distorted genitalia.

L.O., aged 51, was a well-developed, healthy white male. Physical abnormalities centred entirely about his external configuration. Both breasts were grossly scarred and pendulous, with areas of rock-like consistency. The nipples were distorted. The lower abdominal wall was formed by two large panniculi similarly distorted and scarred. The glans penis was normal, but the shaft was fully three times the normal diameter and subcutaneous masses were palpable. The scrotum was similarly involved. Both testicles were palpable and responded to stimuli. He denied loss of libido or capacity for orgasm.



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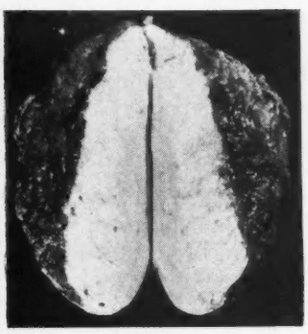
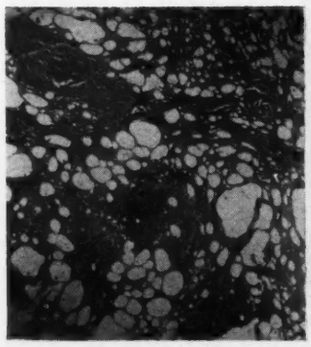


Fig. 4.

Figs. 3 and 4.—Cut surfaces of breasts containing free oil in saccules surrounded by dense fibrous tissue. Combined weight of breasts was in excess of 8 lb.



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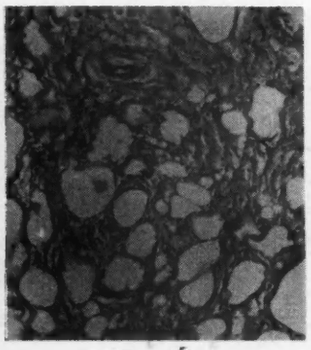

Fig. 6.

Figs. 5 and 6.—Microscopically, the oil vacuoles are seen with lipophages surrounded by giant cells and the connecting stroma of fibrous tissue.

The first surgical procedure involved bilateral mastectomies. This was technically difficult since the granulomatous areas had invaded the intercostal muscles in some areas, and bleeding was not easily controlled. However, it was possible to approximate sufficient healthy skin, and no grafting was necessary. At operation, free oil, subsequently shown to be vegetable in nature, was recovered from the interior of the granulomatous capsule.

The second procedure consisted of reconstruction of his genitalia. The prepuce was freed as well as subcutaneous tissue containing the granuloma from the shaft of the penis to the depth of Buck's fascia. None of the skin covering the shaft could be used for recovering the organ. With a ventral incision in the midline, it was removed completely, along with the skin and subcutaneous tissue of the anterior surface of the scrotum which was involved in a similar process. Finally, to repair the defect necessary to cover the shaft of the penis, the latter was embedded in the scrotum. The granulomatous reaction appeared to be outside of Buck's fascia, except for a small portion which was adherent ventrally (Figs. 1-6).

Acknowledgments are due Dr. D. L. Roxburgh, pathologist, St. Joseph's Hospital, Dr. John A. Hopkins, urologist, and Mr. L. Spencer, who prepared the photographs.

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PAGES OUT OF THE PAST: FROM THE JOURNAL OF FIFTY YEARS AGO

STYLE IN MEDICAL WRITING

Many writers appear to think that a good style may be achieved by the use of flamboyant language. The following will serve as an illustration: "Life is a biologic function. Living matter has a definite chemic composition, wasting by oxidation and reintegrating by the assimilation of new matter. The specific feature of living matter is its chemic mutability, and which, *mutatis mutandis*, is a history of food changes plus the mechanisms by which its potential energy is converted into vital force." Or, again, "the disequilibration associated with the cessation of ovulation and menstruation is a menace to mental integrity." Also, there are many phrases which, although quite proper in themselves, have become worn and threadbare by constant use, and their continual employment destroys all sense of freshness. Such expressions as "etiological factor", "pathological findings", "diagnostic significance", "clinical picture", "symptom-complex", were once good; but they have long since fallen from their high estate. A careful comparison of the older issues of medical journals with the newer ones forces one to the melancholy conclusion that, in respect of style, the old were better than the new. The fault is largely with the younger writers.—Editorial, *Canadian Medical Association Journal*, 1: 72, January 1911.

Canad. M. A. J.
Jan. 28, 1961, vol. 84

RECURRENT DISLOCATION OF THE ELBOW

PAUL McGOEY, *Toronto*

Miss M.M., a schoolgirl, was first seen on May 14, 1956, at the age of 13 years. At age 9 she sustained a posterior dislocation of the left elbow (Fig. 1 A). This was reduced under general anesthesia and immobilized in plaster. During the intervening four years, she had approximately 25 recurrent dislocations. Shortly before her first visit the elbow had been dislocating with increasing frequency. Recently, she had been able to reduce the dislocations and continue her activities with only slight discomfort for several hours.

On May 22, 1956, an operation was performed at St. Michael's Hospital, Toronto. With the patient under anesthesia, it was observed that whenever the elbow was extended through the final 45°, a posterior dislocation occurred. In addition, there was gross lateral instability. The arm tilted readily into valgus and there was no sign of a medial ligament.

Because of the latter condition we felt that it would be worth while to reconstruct the medial ligament in addition to placing a bone graft in the region of the coronoid process. Through a short medial incision the ulnar nerve was exposed. A medial ligament was reconstructed with autogenous fascia lata. A block of iliac bone was taken and placed in the region of the coronoid process as illustrated in the x-ray film (Fig. 1 B). A plaster was applied for three weeks.

Since that time the child has progressed very well. The final examination on January 9, 1959, revealed a full range of movement as shown in the radiographs (Figs. 1 C and 1 D). Apart from occasional mild discomfort in damp weather the patient enjoys excellent function.

DISCUSSION

Recurrent dislocations of the elbow are very rare.

The literature on this subject has been reviewed by King[1] and von Stapelmohr.[2] The variety of pathological findings is interesting, as are the numerous corrective operations which have been described. The presence of fractures, rupture of the capsule or brachialis muscle, injuries to the lateral ligaments, osteochondritis dissecans with loose bodies, a shallow trochlear notch with a deficient coronoid process have all been described. Operations to increase the size of the trochlear notch by an anterior bone block or to repair defects of the capsule and ligaments have been described.[1, 2] A transfer of the biceps tendon to the coronoid process has been used successfully.[3]

In this patient, a shallow trochlear notch due to a poorly developed coronoid process and a ruptured medial ligament were the two primary abnormalities. An iliac bone block was placed over the coronoid process and the medial ligament was reconstructed with a strip of fascia lata. The short incision over the inner surface of the elbow is scarcely noticeable. The patient has an excellent result from this relatively simple procedure.

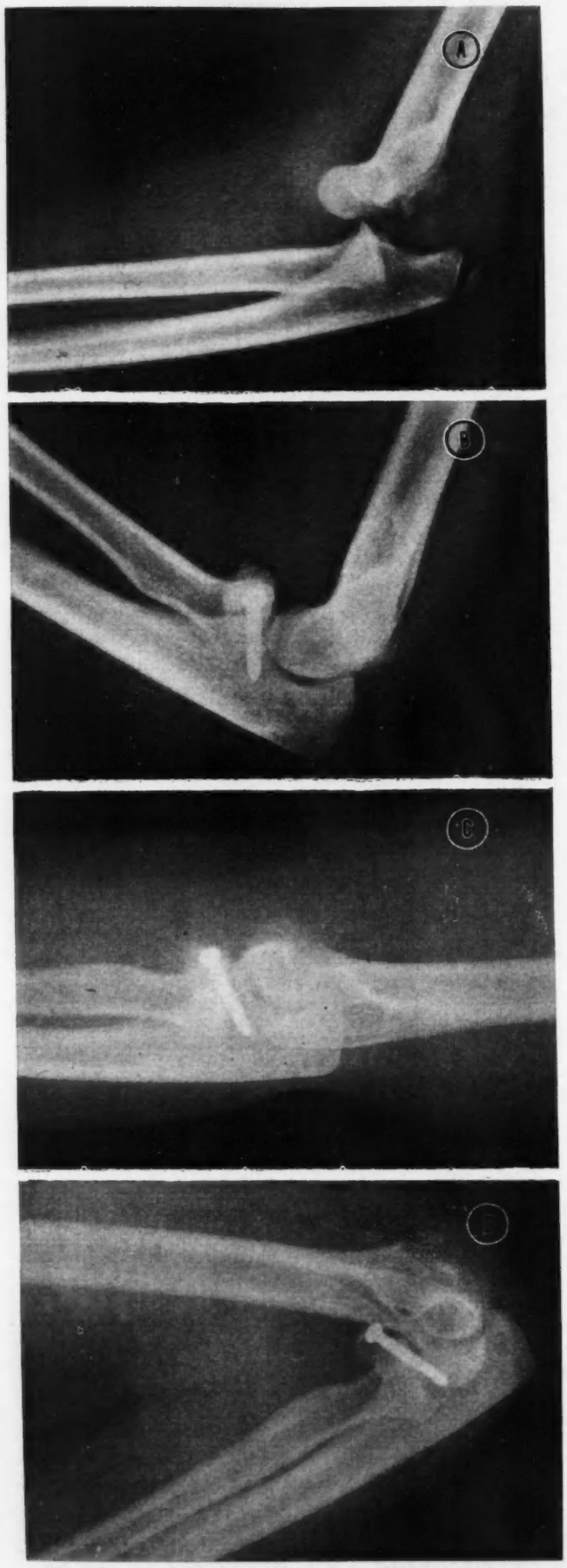

Fig. 1

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MUCOID CARCINOMA ARISING IN A FISTULA-IN-ANO

B. J. LARSON, M.D., J. W. HUNT, M.D. and A. R. BAINBOROUGH, M.D., *Lethbridge, Alta.*

"CARCINOMA IS a rare complication of anal fistula"[1] and perhaps because of its rarity, its possibility is not often entertained when considering the nature of a perianal mass. The following case is therefore presented.

Mr. P.C., aged 43 years, had suffered from a right-sided fistula-in-ano for 12 years, which periodically drained a copious amount of pus. During the last three years, the discharge had been jelly-like and in the last year a mass had formed which enlarged slowly to a size of two inches in diameter and had become indurated. It was located to the right of, and posterior to, the anus. The past history otherwise contributed nothing towards eliciting the nature of the lesion, which was finally excised along with overlying skin and surrounding tissue down to the external anal sphincter.

The mass, when sectioned, proved to be a multilocular cyst containing gelatinous material. Microscopically the cystlike spaces were found to be lined by mucus-secreting cells, among which were numerous mitotic figures. In the mucus content of the spaces were small clumps of well-preserved, desquamated epithelial cells (Fig. 1). It was considered at first that this was a mucoid carcinoma of the rectum extending into a fistulous tract, but sigmoidoscopic examination performed eight days later failed to reveal a neoplasm in the terminal 15 cm. of large bowel. Barium enema and chest radiograph examinations did not reveal any evidence of a lesion higher in the gastrointestinal tract or of metastatic spread. Therefore an abdominoperineal resection of the rectum was carried out 17 days after resection of the perianal mass. No tumour was found in the surgical specimen, but in the microscopic section of the site of the original mass a few islands of neoplastic cells were found, emphasizing the inadvisability of removing only the tumour in such a case.

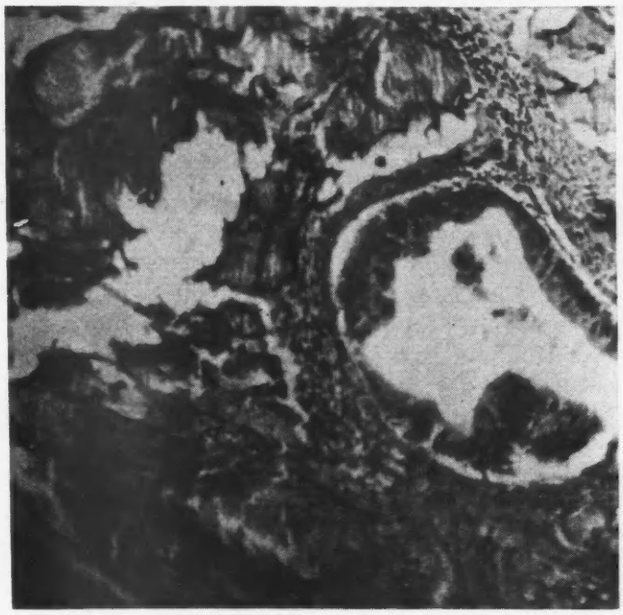

Fig. 1.—Mucoid carcinoma arising in a fistula-in-ano. One gland and spaces containing pools of mucus are shown.

DISCUSSION

In 1931, Rosser[2] collected from the literature seven cases of carcinoma arising in a fistula-in-ano and added seven of his own. Since that time ten more have been reported.[3, 4] Of these 24, one of Rosser's (Case 7) may not be acceptable as a true example of carcinoma arising in a fistula-in-ano. Among the remaining 23, 11 were of the mucoid or colloid type of adenocarcinoma. All of the eight cases of Dukes and Galvin[3] were of this type. These authors reported two further cases, one in a male and one in a female, 71 and 44 years of age, respectively, in which multiple fistulous tracts were found to be lined by mucus-secretory, non-malignant epithelial cells. This led them to believe that in certain individuals there are about the lower rectum and anus congenitally ectopic tracts representing small duplications of the bowel, lined by normal mucus-secreting columnar epithelium, which may on occasion undergo malignant change. Gross[5] describes larger, clinically significant duplications of the rectum in young children. Several reports have been published[6-9] of cases of perianal carcinoma of squamous cell, transitional cell or glandular type which are believed to have arisen from the intramuscular glands, which are present in this area. These glands have ducts lined by squamous or transitional epithelium and end in blind, bulbous expansions, lined by columnar epithelium which is not usually mucus-secretory.

Of the 12 patients with mucoid carcinoma arising in a fistula-in-ano, including the present case, 11 were males. In these cases the fistula had preceded the discovery of carcinoma by one to forty-four years.

This case, as well as the others previously reported, points out the importance of pathological examination of fistulae-in-ano. Many fistulae appear to be adequately treated by incision and drainage, but where the discharge is mucoid, healing prolonged or there is a progressively enlarging perianal mass, carcinoma should be suspected and excision for biopsy should be performed. Hirschmann and Rosenblatt[10] report a case of carcinoma discovered by a routine histological examination of prolapsing hemorrhoids which appeared to be thrombosed. It is apparent, then, that by routine examination of hemorrhoids and other apparently benign anal lesions, more early carcinomas of this region may be found in a stage of growth amenable to surgical extirpation. A review of the literature suggests that such lesions spread via the lymphatics and therefore abdominoperineal resection is more likely to be curative than purely regional excision.

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SHORT COMMUNICATION

THE TONOMETER IN THE EARLY DIAGNOSIS OF GLAUCOMA

S. M. DRANCE, M.B., F.R.C.S.(Eng.),*
Saskatoon, Sask.

THE REALIZATION that glaucoma presents a social problem in the community has increased in the last two decades and has resulted in the establishment of a number of glaucoma clinics from coast to coast in Canada. The disease is insidious, progressive and irreversibly damaging to the visual organ, causing 10 to 15% of all blindness in this country and an even larger percentage of visual disability not amounting to blindness. The figure of 2 to 2½% is quoted from surveys in different parts of the world as the incidence of chronic simple glaucoma in the population over the age of 40 years. Early diagnosis of the disease, probably the only means by which one can prevent irreversible changes of the optic nerve head and visual field, should therefore be the aim of general practitioners, ophthalmologists and optometrists, rather than the treatment of very late and hopeless cases. One should suspect the disease whenever there is (1) a family history of glaucoma, (2) premature onset of presbyopia, (3) a history of frequent change of spectacles, (4) elevated intraocular pressure on a routine examination, or (5) one or other symptoms of ocular discomfort, grittiness and possibly headache, particularly on waking in the morning, and (6) in all patients over the age of 40.

A number of authors have stressed that the best method of early diagnosis is by the routine use of the tonometer when recording the intraocular pressure of patients undergoing an annual medical examination, an insurance examination or routine refraction. Reed[1] has reported some valuable studies on the routine use of the tonometer in patients over the age of 40. He found that of 5000 patients examined, early glaucoma was detected by the routine use of the tonometer in 43, while advanced glaucoma was clinically detectable by other means in another 43.

The tonometer commonly used is the Schiötz tonometer, which measures the intraocular pressure by indenting the cornea with a fixed weight. This instrument is easy to use and the procedure is not time-consuming. Therefore the measurement can be easily carried out at the end of a physical examination. A raised value for intraocular pressure obtained by the Schiötz tonometer almost always indicates the presence of glaucoma, but it is important to be aware of the limitations of an isolated reading and of some of the pitfalls of the Schiötz tonometer so that a reading can be correctly interpreted.

The first and probably the most important source of error in relying on an isolated tonometric reading lies in the fact that the intraocular pressure shows diurnal variations analogous to most of the other vegetative functions. This diurnal variation in pressure has been recognized since the early part of this century, and many authors believe that the first deviation from normal is the inability to maintain the intraocular pressure variations within minor limits. Intraocular pressure therefore becomes labile before showing "pathologically elevated pressures". A recent analysis[2] of the significance of the diurnal phasic tension variations in normal and glaucomatous eyes revealed the phasic variation of intraocular pressure to be less than 5 mm. of Hg in 84% of normal eyes. In 42% of these eyes the maximum intraocular pressure was at 6 o'clock in the morning, on waking and before the patient was up (the converse is true of arterial blood pressure).

In untreated eyes with chronic simple glaucoma only 6% showed phasic variation of intraocular pressure of less than 5 mm. of Hg and in some the swings of intraocular pressure were more than 25 mm. in the 24 hours. In 60% of these eyes the intraocular pressure was maximal during the usual non-office hours and in only 40% was the peak value reached during office hours. Only 12% of the glaucomatous eyes examined had a pressure which was consistently elevated above 25 mm. of Hg throughout the 24 hours of the night and day; only

*Director of the Glaucoma Clinic, University Hospital, Saskatoon.

these patients would be detected by an isolated Schiötz tonometer examination irrespective of the time of examination. The significance of this lies in the fact that when chronic simple glaucoma is suspected a normal intraocular pressure does not exclude the disease, as the patient may show considerably elevated intraocular pressures at times when the intraocular pressures can be checked only if the patient is in hospital. An understanding of these considerations would go a long way in promoting the enlightened and proper use of the Schiötz tonometer.

Impression tonometry, including Schiötz tonometry, is influenced by the rigidity of the coats of the eye and the radius of curvature of the cornea. The radius of curvature of corneae, apart from congenital malformation and buphthalmos, usually shows little deviation from the mean. Scleral rigidity as a cause, however, has recently received a great deal of study.[3-6] In eyes with soft sclerae the weight of the Schiötz tonometer produces more indentation than it would in eyes with normal or high scleral rigidity. The conversion tables are, however, worked out for a normal mean scleral rigidity and will therefore give erroneously low readings of intraocular pressure. In short-sighted patients the sclera is usually soft and this source of error becomes important, as the intraocular pressure may be elevated even though the Schiötz tonometer shows normal readings. This probably accounts for the notion that glaucoma is rare in short-sighted people. Modern methods of examination suggest that this is not true. Conversely, high scleral rigidity accounts for higher intraocular pressure readings than are actually present in the eye and results in a mistaken diagnosis of glaucoma, which accounts for some of the so-called nonprogressive cases.

The final pitfall in tonometry that should be mentioned occurs in the diagnosis of angle closure glaucoma or congestive glaucoma. Patients with this disease have very shallow anterior chambers and are subject to sudden rises of intraocular pressure with corneal edema, pain, redness and blurring of vision with characteristic rainbows. In the early stages when the attacks of glaucoma are mild and disappear after a satisfactory night's sleep the intraocular pressures may be completely normal in the intervals between the attacks. A history of blurring of vision and coloured rings around lights must not be dismissed because of a normal intraocular pressure recording in an interval period. This is particularly important because of the good prognosis of this disease if surgical treatment is carried out early.

SUMMARY

Routine use of the Schiötz tonometer in people over 40 years of age will bring to light very early cases of glaucoma and will result in a better prognosis. As an aid to making a correct interpretation of the tonometric reading the common causes of error in interpretation have been outlined. Two of these arise from the nature of the disease and one from the method of tonometry itself. A raised intraocular pressure almost always means glaucoma. A normal reading, though reassuring, does not exclude glaucoma.

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PAGES OUT OF THE PAST: FROM THE JOURNAL OF FIFTY YEARS AGO

CAISSON-DISEASE IN CANADA

The rapid growth of bridge construction and tunnel work in Canada is directing attention to the cause and treatment of caisson-disease. We note in the November number of the *Maritime Medical News*, an article on the "Etiology of Caisson-Disease", by Dr. P. Conroy, of Charlottetown. Dr. Conroy, for three years, was medical attendant to several hundred caisson workers employed in the construction of the Hillsborough bridge, at Charlottetown, and as the caissons for that structure were sunk to a depth requiring, at times, more than three pressures of atmosphere, he had an opportunity to study carefully this malady.

One of the most interesting points brought out in Dr. Conroy's paper is the fact that the caisson workers have learned by experience the best method of treatment, and will not brook any interference on the part of the doctor. When a workman develops the disease, he is quickly taken charge of by his comrades, and placed in a hot bath. A course of vigorous rubbing is begun, and kept up until the patient has recovered, or until further treatment is deemed unavailing. The convalescent is then kept quiet in a hot room for several hours.

His study of the conditions under which the men work, and the order in which symptoms of the malady develop, have convinced Dr. Conroy that caisson-disease is a toxemia due to excessive catabolism. The facts which he cites are interesting, not only as in support of his conclusion, but as throwing light upon the peculiar conditions under which work in caissons is carried on.—*Canadian Medical Association Journal*, 1: 73, January 1911.

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DOCTORS AND DEATH

DESPITE our best efforts we can only stay death by a few decades, a few years, or for an even shorter time. All of our patients die eventually, but as the unknown Yankee soldier put it, "the difference between dying today and dying tomorrow is not much, but we all prefer tomorrow". Shakespeare put it another way, "Be absolute for death, either death or life will thereby be the sweeter." Perhaps we are becoming more squeamish about death than our medical forebears for whom it was a frequent and inevitable happening. Death is undoubtedly becoming cleaner, more anonymous and less painful. It is indeed in danger of developing a certain horrific and fatuous hygienity, such as Aldous Huxley depicted in his "Brave New World". When I visited Forest Lawns, the great cemetery in Los Angeles, I was told of a sardonic visitor who commented as he surveyed those broad acres filled with bad statuary, sentiment and soft music, exuding an atmosphere of general syrupy cheerfulness, "Death, here is thy sting!" Death and dying cannot be shrugged off or put to one side. As medical men we cannot evade the issues, and indeed we seldom attempt to do so, but not infrequently we pass by a little to the other side. Awareness of and preoccupation with death has played a great part in human affairs; some of our finest buildings, from the pyramids to the Taj Mahal, have been inspired by death, and so have many of the greatest poems and epics. Whole sciences have developed in attempts to delay, further, or forestall it. Death is inescapable and there are never any easy answers for it. The busy doctor works out some rules for himself over the years, and with the common sense which his kind usually shows, he probably makes fewer mistakes than most. The family doctor is, or certainly should be, best able to tell the patient and his family that death is coming.

A recent translation by Rosemary Edmonds of Leo Tolstoy's "The Death of Ivan Ilyich"[1] is admirable reading for those who would explore the psychology of dying through the eyes and heart of a great writer. There are only 60 pages and it can be read in an evening. Ivan Ilyich is a Russian civil servant, a lawyer, who becomes ill. Tolstoy sets the stage with macabre skill as he records this conversation at Ivan Ilyich's funeral, between his wife Praskovya Fiodorovna and his friend Piotr Ivanovitch. "The last few days of his suffering were dreadful." "He suffered much?" asked Piotr Ivanovitch. "Oh, dreadfully. At the last he screamed not for minutes but for hours on end. During three days and nights he screamed incessantly. It was unendurable. I don't know how I bore it. You could hear him three rooms away. Oh, what I have gone through!"

After a sketch of Ivan Ilyich's life we are brought to his illness. Tolstoy compares the rituals of medicine with those of the law; "the entire procedure was just the same as in the law courts. The airs that he put on in the courts for the benefit of the prisoners at the bar, the doctors now put on for him . . . The doctor summed up just as brilliantly, looking over his spectacles triumphantly, gaily even, at the accused. From the doctor's summing up Ivan Ilyich concluded that things looked bad, but that for the doctor and most likely for everyone else it was a matter of indifference, though for him it was bad." The illness progresses and Tolstoy maps its course. Ivan Ilyich has what sounds like a cancer of the bowel. In the 1860's operations were rarely undertaken, even *in extremis*. Gradually it dawns on Ivan Ilyich that "he was dying and he was in continual despair. In the depths of his heart he knew that he was dying, but so far from getting used to the idea he simply did not and could not grasp it." But although he knows this, and he knows that his family know this too, none of them can admit it. "And it was a strange thing, many a time when they were playing their farce for his benefit he was within a hair breadth of shouting at them, 'Stop lying, you know and I know that I am dying.' But he had never had the spirit to do it. The awful, terrible act of his dying was reduced by those around him to the level of a fortuitous, disagreeable and rather indecent incident." Only Gerassim, his peasant serving man, was honest with him, "Gerassim alone told no lies." Everything showed that he alone understood the facts of the case and did not consider it necessary to disguise them, and simply felt sorry for his sick, expiring master. On one occasion when Ivan Ilyich was for sending him away to bed, he even said straight out, "We shall all of us die, so what's a little trouble." . . . "Apart from this lying, or in consequence of it, the most wretched thing for Ivan Ilyich was that nobody pitied him as he yearned to be pitied. At certain moments after a prolonged bout of suffering, he craved more than any-

thing—ashamed as he would have been to own it—for someone to feel sorry for him as if he were a sick child. He longed to be petted, kissed and wept over as children are petted over . . . and in Gerassim's attitude towards him there was something akin to what he yearned for, and so Gerassim was a comfort to him . . . The falsity around him and within him did more than anything else to poison Ivan Ilyich's last days." It is a terrible story, and the translation is excellent.

Have we gone far beyond the pain and falsity of Ivan Ilyich's days nearly a century ago? Certainly we have much more effective treatment and not so much humbug is needed, for the wretched Ivan Ilyich was handed from doctor to doctor, and his pain was compounded by dread and terror. But are we using this opportunity to meet death with dignity, resolve and understanding, for it is one of the great Universals which cannot be sidestepped? Or are we escaping from the agonized falsity of Ivan Ilyich to Aldous Huxley's appalling Park Lane Hospital for the Dying with its sixty-storey tower of primrose-coloured tiles and its bevy of gaily coloured aerial hearses where the air was continuously alive with gay synthetic melodies? At the foot of every bed confronting its moribund occupant was a television box. Television was left on a running tap from morning till night. Every quarter of an hour the prevailing perfume of the room was automatically changed. "We try," explained the nurse, who had taken charge of the savage at the door, "we try to create a thoroughly pleasant atmosphere here—something between a first-class hotel and a feely palace—if you take my meaning." The brave new worlders die to the sobbing crescendo of the super vox Wurlitziana and the scent of verbena and patchouli wafting through. And they are watched by "five khaki twins, each with the stump of a long eclair in his right hand, and their identical faces smeared with liquid chocolate", who are being given their "wholesome death conditioning".

Mr. Huxley has been too accurate a prophet for us to ignore his warning. The brave new worlders may have had a more comfortable ending than Ivan Ilyich's three days of screaming, but is this really the best that we can do? Medical science has extended life and made our departure less agonizing though often more prolonged than it used to be. As medical men we should surely ponder these matters, remembering the words of Queen Elizabeth the First in her petition to her seamen, that "the wings of man's life are plumed by the feathers of death". H.O.

REFERENCE

1. TOLSTOY, L.: The Cossacks, and the death of Ivan Ilyich, a new translation by Rosemary Edmonds, Penguin Classics, London, 1960.

PILLS FOR BIRTH CONTROL

CONSIDERABLE interest has been engendered by the lay press about the extensive possibilities of contraception by the simple expedient of taking one small tablet a day. Physicians are frequently being asked about the possibilities and use of these tablets which will dispense with all mechanical methods.

There are now available several new progestational steroids for oral and parenteral use. The newer 19-Nor-steroid compounds appear to be beneficial in cases of dysfunctional menstrual disorders, habitual abortions and sterility due to an inadequate progestational phase. There is good reason to believe that for short-term therapy of these specific gynecological conditions, these substances are highly effective. The microscopic picture of the endometrium produced by these compounds has been described as a peculiar and unique response in the form of a pronounced decidua-like reaction with atrophy of the glands. However, natural progesterone produces a similar picture and this appears to be a characteristic response over a longer period of time. It may be stated, therefore, that these newer substances imitate the natural action of the corpus luteum as no other preparation hitherto has been capable of doing. This finding naturally pertains to the management of those conditions which are alleviated by suppression of ovulation, i.e., dysmenorrhea, endometriosis and fertility.

The fundamental aim of birth control obviously is to limit the number of pregnancies. With these 19-Nor-steroid compounds, the mechanism is one of suppression of the gonadotropins from the anterior pituitary with direct inhibition of ovulation. The primary requisites of any contraceptive are effectiveness, simplicity, economy, safety and a minimum of unpleasant side effects. These compounds, when used in a cyclic fashion from day five through day 24 of the menstrual cycle, unquestionably are effective in suppression of ovulation. The only failures appear to be a direct result of patient neglect or disuse. The compounds are obviously simple to use with reasonable care.

Economically, the mechanical methods of birth control are superior, but it appears that with discrete purchasing, a minimum 2.5 g. daily dose for 20 days would cost between $2.00 and $3.00 per month. It is anticipated that even this expense may be reduced with changes in production methods.

About 20% of the women involved in reported trials of the 19-Nor-steroids discontinued their use because of undesirable side effects. Evidence suggests that once discontinuance of these compounds is made, there is a prompt return of ovulation. There is still considerable hesitation on the part of any investigator to state dogmatically that there are no long-term harmful effects arising from the protracted use of these compounds for birth control. W.F.B.

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THE EVOLUTION OF INTERNATIONAL COMMUNICATION IN MEDICAL SCIENCES

FOR centuries following the birth of science, its international nature was reflected solely by personal visits, correspondence and exchange of views between individual scientists working in different centres and laboratories in many countries. These were the only channels of communication by means of which the results of medical research and clinical observation could be disseminated. Later the exchange of scientific information was intensified with the advent of an increasing number of journals and specialized publications.

The past century has witnessed the development of an ever-expanding network of international organizations concerned with problems peculiar to specific and increasingly specialized areas of medicine. These organizations have fostered a growing number of international congresses at which scientists from all countries gather to acquire information from one another and to draw inspiration for their subsequent endeavours.

Though their objectives are admirable and they undoubtedly serve a most useful function, the international medical congresses are in potential danger of getting out of hand, by their very multiplicity, frequency, size and inevitably overlapping scope. The publication of their proceedings, however prompt, often provides the research worker with little more than a picture of the past. In addition it is an expensive proposition requiring of many physicians a great deal of time and effort which they can ill afford. This situation has prompted one organization concerned with promotion of international scientific communication to observe pessimistically that we are making progress in reverse and that "in this era of the electronic computer we are back to the bush telegraph".

Some years ago a body known as the Council for International Organizations of Medical Sciences (CIOMS) was founded under the auspices of the World Health Organization and UNESCO.

Among its functions, CIOMS acts as a source of information, co-ordination and assistance to its member international medical organizations with particular reference to the planning of international congresses of all types.

One of the most recent developments in the network of scientific communication is a function designated as the "international symposium", in the promotion of which CIOMS has played an active role. These international symposia are carefully planned meetings of several days' duration which bring together about 15 to 25 leading investigators belonging to several disciplines. To economize on the time required to impart factual information, the papers on the agenda are distributed to all members before the meeting. To promote a sense of unity and to improve occasions for discussion, common housing and meal facilities are provided for the participants. To ensure a maximum of freedom of expression, no audience is invited as a rule.

CIOMS considers that these symposia constitute the most productive type of international scientific meetings but recognizes that they have many inherent limitations. They have proved of considerable value when a subject is progressing rapidly and it becomes necessary to take stock, to evaluate and integrate newly acquired data. They may also be of use when progress in a particular problem appears to be stagnating despite a large volume of research and it becomes necessary to discover ways and means to circumvent such stalemates.

Nevertheless, their ultimate value suffers by the fact that they are often enshrouded in secrecy and because by their very nature they must be held on a "by invitation only" basis without advertisement to the general medical public. A major limitation is inherent in the restricted size of the audience and the necessarily arbitrary choice of participants. It is impossible to invite all those who may have made significant contributions to any particular subject, and though attempts may be made to include investigators of varying seniority, usually only a small number of younger people can attend these international symposia, a situation leading to much unavoidable frustration. To some extent these drawbacks may be mitigated by publication of the proceedings of such symposia but their editing is a lengthy business and classical methods of publication require many weeks at best. Delay in return of proofs by a single delinquent contributor can hold up the entire process. There is much to be said for the practice of organizing these international meetings in such a way that the papers and discussions are edited by participants before they disperse. There is also a need for development and use of faster methods of publication.

A further offshoot of the formal international symposium, even narrower in scope, is the very small and informal meeting of six or eight leading scientists with the purpose of studying a well-circumscribed problem. Such "brain-storming" sessions require no elaborate preparation, no prepared papers and no verbatim proceedings. Conclusions may readily take the form of a short report to a scientific journal.

With clearer realization of the day-to-day extension of the fields of investigation in both the science and practice of medicine, there is an increasing awareness of the depth of each problem and of the multiplicity of new problems. Though the division of research groups and the need for special knowledge may lead to a greater number of small meetings of an interdisciplinary nature, there is a potential danger that much time may be wasted by scientists playing "an endless peripatetic party game of musical chairs", reading the same papers each time the music stops.

LETTERS TO THE EDITOR

TO EXCHANGE OR NOT TO EXCHANGE

To the Editor:

I am in agreement with Dr. E. J. Turner (*Canad. M. A. J.*, 83: 1387, 1960) about not necessarily performing an exchange transfusion immediately the indirect bilirubin reaches 20 mg. %.

In such cases of Rh, ABO or AB-A or B incompatibility, there is one laboratory test which I have found very useful, namely the reticulocyte count. Here are some personal impressions from a somewhat limited experience:

1. A reticulocyte count of 10% or more on cord blood usually means very serious disease, with eventual need for an exchange transfusion.

2. A reticulocyte count of 6% on cord blood is suggestive of moderate disease, where the indirect serum bilirubin level should probably approach the 20 mg. % mark, but where exchange transfusion will probably not be required.

3. The reticulocyte count should be repeated as often as is the serum bilirubin determination. When, in the two or three days immediately after a transfusion, the indirect bilirubin increases towards the 20 mg. % mark, but where the reticulocyte count at the same time *decreases*, there will usually be no need for a second exchange transfusion.

GILLES MARION, M.D., C.M., F.A.A.P.,
168 Charlotte St.,
Ottawa, Ontario.

THE CONTINUING CHALLENGE OF BREAST CANCER

To the Editor:

In this journal (83: 1229-1233, December 10, 1960) Dr. Ivan H. Smith says, "Fortunately, in this country his [McKinnon's] deductions regarding the importance of early diagnosis in prognosis [in breast cancer] have been brushed aside, for optimism and service are not usually willing to wait because of a statistical interpretation." I will not impose on this space with further evidence of the validity of the deductions except to mention a recent report by Simmons S. Smith, M.D., and Alfred C. Meyer, M.D., in the *American Journal of Surgery*, 98: 653, 1959. In this entirely objective survey of treated patients they found, *contrary to their expectations*, that "Neither the 5- nor the 10-year survival rates were affected by the type of operation, the duration of the tumour, or the skill of the surgeon."

But, in fairness to Dr. Delarue and Dr. Janes, I must refer Dr. Smith to this journal (78: 781, 1958) wherein they are cited, and also to Dr. Janes, "Carcinoma of the breast: a reassessment", in the *Canadian Journal of Surgery*, 2: 252, 1959. He will find there that the deductions have not been so entirely "brushed aside", even in this country, and that his use of the word "fortunately" is rather unfortunate.

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MEDICAL NEWS IN BRIEF

CHROMOSOME NUMBERS IN CELLS CULTURED FROM HUMAN SYNOVIAL TISSUE

Techniques for enumerating chromosomes in cultured cells provide an opportunity to "monitor" cells derived from fresh tissue for evidence of aberration from normal. Since chromosomal patterns may be significant in tissue regeneration, such studies could provide information in the self-perpetuating chronic inflammatory diseases. Castor (*Arthritis & Rheumat.*, 3: 436, 1960) conducted such studies on synovial cells from four subjects with microscopically normal synovium (two of whom had systemic lupus erythematosus and two had no rheumatic disease) and three patients with rheumatoid arthritis who exhibited clinical and microscopic evidence of chronic synovitis.

In cells cultured from persons with normal synovium, deviations from the normal human diploid chromosome complement ($2n = 46$) occurred as early as the second subculture. Aberrant forms in early cultures were predominantly hypodiploid (40 to 45 chromosomes per cell), while later subcultures exhibited hypotetraploid and polyploid counts. In two persons in whom counts were performed in early cellular derivatives, normal diploid cells accounted for 57% and 58%, respectively, of the metaphase plates counted in the second subculture. Cells from one of these individuals still exhibited 62% normal diploid counts at the third subculture, and in a third patient, 59% of cells examined at the twenty-first subculture had the normal diploid chromosome number.

Early cultures from those with rheumatoid arthritis revealed the normal diploid number in 31, 28 and 46% of the cells examined from the three patients. A wide range of hypodiploid, hypotetraploid and polyploid counts were present.

Further study will be necessary to determine the reproducibility of these findings and their significance as a reflection of a possible chromosomal defect in rheumatoid synovium.

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STATEMENT ON DIHYDROSTREPTOMYCIN: THE COMMITTEE ON THERAPY, AMERICAN THORACIC SOCIETY

The Committee on Therapy of the American Thoracic Society has issued the following opinion and recommendations concerning the use of dihydrostreptomycin for the treatment of tuberculosis (*Am. Rev. Respiratory Dis.*, 82: 750, 1960). While streptomycin, the parent drug from which dihydrostreptomycin is derived, occasionally produces vestibular damage and deafness, this complication occurs after relatively large doses and does not progress after the drug is discontinued. In contrast, deafness may occur after small doses of dihydrostreptomycin and may progress or even begin after this antibiotic is discontinued. Once developed, hearing loss is usually irreversible and may be characterized by a selective loss of speech discrimination which is difficult to treat. Although serious vestibular damage does not often occur, its irreversibility, the unpredictability of the drug in producing hearing loss, and the difficulty in recognizing the toxic effects early are cited by the committee as reasons for not using dihydrostreptomycin in the routine treatment of common bacillary infections and for not combining it with other antimicrobial agents in commercial preparations.

Since streptomycin alone, when employed in the usual antituberculosis dosage not in excess of one gram daily, has not been associated with the high degree of vestibular damage, and since there is no definite proof of the superiority of regimens which include dihydrostreptomycin, the use of mixtures of streptomycin and dihydrostreptomycin, or dihydrostreptomycin alone, is not recommended for the routine therapy of tuberculosis.

The committee suggests that the use of dihydrostreptomycin in tuberculosis should be limited to rare cases in which there is a specific hypersensitivity to streptomycin. In such circumstances where the indications are compelling, the committee feels that the risk of deafness from dihydrostreptomycin should not absolutely preclude its use. Therefore the continuing availability of this drug for use in such circumstances is desirable.

ESTROGENS AND HEMOSTASIS

During recent years, Premarin®, a complex of estrogen sulfates prepared from the urine of pregnant mares, has been widely advocated and used as a hemostatic agent, mainly for the control of epistaxis but also to control bleeding during and after certain surgical procedures, particularly those involving the prostate gland. In 1941 Jacobson (*Virginia M. Month.*, 68: 37, 1941) advanced the concept of a clinical entity which he termed "spontaneous bleeding" to apply to bleeding without recognizable cause in the form of any type of blood dyscrasia, trauma, operation, ulcer, neoplasm or the like. He postulated that this entity was caused by low blood levels of estrogen and proposed that such bleeding might be arrested by the administration of Premarin. Experimental support of this concept was reported in 1957 by Johnson (*Proc. Soc. Exper. Biol.*, 94: 92, 1957), who injected Premarin into 12 dogs and 15 to 30 minutes later detected an increase in prothrombin and proaccelerin (factor V), and a decrease of antithrombin, the effect of the drug lasting several hours. He concluded that an elevation of proaccelerin might be of value in control and/or prevention of bleeding, should it be shown that this effect of estrogen upon dogs also applies to humans.

In the past few years several clinical reports have been published claiming favourable effects from the use of Premarin as a hemostatic agent without deleterious side effects. The types of bleeding said to have been controlled by this drug include epistaxis, post-tonsillectomy hemorrhage, rectal and gastrointestinal bleeding, subarachnoid hemorrhage, and bleeding occurring during various operative procedures.

On the basis that the theoretical concept of "spontaneous bleeding" was unsound, that laboratory data reported to date could hardly explain the clinical effects that have been claimed, and that all the clinical studies claiming beneficial effects from this drug lacked adequate control groups, Borchgrevink, Andersen, Hall, Hatteland and Ursin-Holm of Oslo (*Brit. M. J.*, 2: 1645, 1960) undertook a controlled study of the effects of intravenous injection of 20 mg. of Premarin. This investigation included observations on a battery of laboratory tests of the various coagulation factors, bleeding time and tourniquet test in four normal women and four normal men, on the effects of the drug on post-prostatectomy blood loss (31 patients), and on the comparative effects of intravenous Premarin and a placebo on 11 patients with severe "spontaneous" epistaxis.

These authors concluded that intravenously administered Premarin in doses of 20 mg. has no demonstrable effect on various vascular, platelet and coagulation functions in normal individuals; nor upon prolonged bleeding time or positive tourniquet tests in patients with hemorrhagic diatheses; nor upon blood loss following prostatectomy, or epistaxis. In those with epistaxis, a placebo effect only could be demonstrated. No undesirable side effects were observed in this study. The authors could elicit no evidence to indicate that the previously postulated "low blood level of estrogen" actually exists or results in "spontaneous bleeding".

TOXIN-ANTITOXIN PHENOMENON IN BURNS

Rosenthal, Hartney and Spurrier (*J. A. M. A.*, 174: 957, 1960) have indicated the presence of toxins in the blood of burned or otherwise injured patients by the demonstration of cytotoxic effects on HeLa cells, cytolytic effects on red cells of other burned or injured patients, and precipitogens against sera from patients with healed burns. Such sera were shown to contain antitoxic-antibody-like substances by demonstrating their ability to neutralize the cytotoxic effect on HeLa cells and by demonstrating the presence of hemolysins and precipitins.

In preliminary clinical trials the transfusion of blood or plasma from donors with healed burns to critically toxic burned or injured patients was followed by prompt improvement. Sera obtained from the recipients after such transfusions were no longer toxic to tissue cultures. Further clinical trials of antisera of known titre from persons with healed burns are recommended.

(Continued on advertising page 19)

Medical News from Parliament

The important medical news from Parliament immediately prior to the Christmas adjournment was the Prime Minister's announcement of the setting up of a Royal Commission on Medical Health Insurance. This comes at a time when there has been much discussion on the subject both within the profession and outside it. It was pleasing to hear that this had come about partly as a result of representations by the Canadian Medical Association. This is good public relations. Further opportunities to improve the public relations of our profession will come when various groups appear before the Commission. The Commission members and their terms of reference will be announced early in the new year.

Earlier in December the House, during a private members' day, debated the problems of mental health. The resolution brought in by Allan Macnaughton (Mont-Royal) asked that the Government consider the advisability of co-operating with provincial authorities and such professional and other groups as may be interested in making a national survey of the extent of mental illness, its causes, problems, and methods of treatment. What made the debate a little out of the ordinary was that the resolution was accepted by the Government (a rare occurrence in Parliamentary procedure).

In accepting the resolution in principle, the Minister of National Health and Welfare outlined what is now being done and some of the difficulties involved. A major problem was the difficulty of defining mental illness. The question of defining the areas to be surveyed would have to be answered. There would also be the problem of obtaining trained personnel for such a survey without diverting them from essential treatment and research establishments.

In 1948 the Federal Government established a grant of four million dollars for mental health. Today this grant has reached $8.7 million. These grants for the period to 1960 have totalled over $54 million and have been used for research in a number of mental health fields. As well, they provide a stimulus for the establishment of psychiatric units in general hospitals and mental health clinics in various communities.

H. M. HORNER, M.D., M.P.

ASSOCIATION NOTES

EXECUTIVE COMMITTEE MEETING—JANUARY 6 AND 7, 1961

The Executive Committee met at C.M.A. House, Toronto, on January 6 and 7, 1961, under the chairmanship of Dr. Murray Douglas of Windsor, Ontario, Chairman of General Council.

The Royal Commission on Health Services

At its meeting of December 5 and 6, 1960, the Committee on Economics brought down a recommendation to the Executive Committee proposing that the C.M.A. request the Federal Government to establish a Royal Commission or committee to study existing and projected health needs and resources of Canada and to explore methods of ensuring the highest standard of health care for all citizens, with due recognition of the C.M.A. Statement on Medical Services Insurance in the process of such a study. It was further recommended that, should the Executive Committee act on this proposal, l'Association des Médecins de Langue Française du Canada be apprised of this decision and invited to collaborate with the C.M.A. in its implementation. A detailed account of the developments subsequent to this recommendation was published in the December 24, 1960, issue of the Journal in "News and Views on the Economics of Medicine". In brief, on direction from the Executive Committee this proposal was communicated to the Prime Minister, who announced in Parliament on December 21, 1960, his Government's decision to constitute a Royal Commission for this purpose. On the same day, Dr. G. D. W. Cameron, Deputy Minister of National Health, invited the General Secretary to an informal discussion of matters relating to the Prime Minister's announcement. It was decided, in conference with the Chairman of General Council, that the responsibility for such conversation should be shared with an elected officer of The Association. Dr. George Wodehouse, the Honorary Treasurer, therefore accompanied the General Secretary to Ottawa on December 30, 1960, where free and widely ranging informal discussions were held with the Minister of National Health and Welfare, the Deputy Minister of Health, and senior officials in their department. Dr. Wodehouse and the General Secretary expressed the opinion that these conversations had been timely, useful and indicative of good liaison with the Department of National Health and Welfare.

After considerable discussion, the Executive Committee voted its unanimous approval of the actions of the Chairman of General Council and of the Secretariat leading to this approach to the Government of Canada

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and to the discussions with the Minister of National Health and Welfare, his Deputy and officials.

It now becomes important for The Association to consider in detail its relationship to the Royal Commission and to make the necessary preparation for such contacts with the Commission as may be anticipated. In view of the fact that demands involved in preparation and submission of material to the Commission are likely to be extreme both in time and effort of the Secretariat, the Executive Committee and certain other committees, it was agreed that matters pertaining to these activities should be given top priority though this might involve curtailment of certain other functions of these officers and committees. It was also agreed that the preparation of memoranda, reports and studies should be primarily and usually a staff responsibility of the Secretariat and that the latter should be instructed to work in close collaboration with a subcommittee to be known as the Executive Sub-Committee on Health Services, consisting of Drs. McMillan, Rabson and Wodehouse, with the President and Chairman of General Council as members, *ex officio*. This sub-committee is responsible to and will report to the Executive Committee. It will maintain liaison with, but will not be responsible to the Committee on Economics. It was agreed that l'Association des Médecins de Langue Française du Canada should be invited to nominate a representative to the Executive Sub-Committee on Health Services, to have the same capacity as that of the *ex officio* members from the C.M.A. It was decided that if the terms of reference of the Royal Commission permit, the C.M.A. should request that it be empowered to appoint an observer to attend the Commission's hearings, and that such observer should be a member of the Secretariat. It was considered that if such action is in keeping with the conduct of the Royal Commission on Health Services, the submission by the C.M.A. of multiple briefs and appearances dealing separately with various aspects of the Commission's study would be preferable to the submission of a single all-inclusive brief. It was agreed that The Association should offer to provide data and reports on such matters as fall within its special field of knowledge, such as medical needs, resources and quality of medical care. Bearing in mind the possibility that the Commission may hold hearings in each province, the C.M.A. Divisions are encouraged to give early and urgent consideration to the preparation of submissions, within the bounds of C.M.A. Statements of Policy, for presentation to the Commission. Close liaison in these activities should also be maintained with The Association's affiliates. It was decided that a letter from the Executive Committee should be despatched immediately to the newly appointed Chairman of the Royal Commission on Health Services, wishing him well in his appointment, assuring him of the co-operation of the C.M.A. in all matters pertaining to the Commission, offering assistance at any time at his request and intimating that The Association would establish further contact with him after appointment of the full Commission and the announcement of its terms of reference. A letter will also be sent from the Executive Committee to all C.M.A. members, outlining The Association's activities to date concerning the Royal Commission on Health Services and drawing attention to the *C.M.A. Journal* insert, "News and Views on the Economics of Medicine", as a source of future information and reports of developments related to the Royal Commission.

C.M.A. Questionnaire on Health Insurance

By a majority vote the Executive Committee accepted the report of the Committee on Prepaid Medical Care (a) stating that Questions 22, 23 and 24 of the C.M.A. Questionnaire on Health Insurance and the replies submitted in answer to these questions were as valid as those concerning any other portion of the questionnaire, and (b) recommending that the information relating to these questions be made available to each Division in the same way as that relating to the first 21 questions.

Saskatchewan Brief to the Advisory Planning Committee on Medical Care

The lengthy and comprehensive brief prepared by the Saskatchewan Division of the C.M.A. (College of Physicians and Surgeons of Saskatchewan) for presentation before the Advisory Planning Committee on Medical Care of the Province of Saskatchewan had been submitted in advance of the meeting to all members of the Executive Committee for study and consideration. It has also been in the possession of the Saskatchewan Advisory Planning Committee and was scheduled for public hearing before that body in Regina on January 12 and 13, 1961. The Executive Committee passed a vote of congratulation to the Saskatchewan Division on the preparation of this brief, stating that the recommendations set forth therein appear to be in the best interests of the health of the people of Saskatchewan.

Public Relations Consultant

As previously directed by the Executive Committee, representatives of eight public relations organizations have been interviewed by the Secretariat. The submissions and relative merits of these firms are to be studied by the Secretariat with the chairman and nucleus committee of the Committee on Public Relations, who will bring to a future Executive Committee meeting their recommendations concerning the retention of a professional public relations consultant.

Annual Meetings

The President-Elect, Dr. G. W. Halpenny, reported satisfactory progress in arrangements for the scientific component of the 1961 annual meeting. For the first time in some years there will be a scientific exhibit, arrangements for which are under the direction of Dr. C. Pattee of Montreal. Dr. Halpenny reviewed the general outline of the various components of the meeting, which will be described in more detail in subsequent announcements in the Journal.

In response to a letter from Professor Ford Connell of Queen's University, drawing attention to a conflict in the dates for the 1964 annual meeting of the C.M.A. and the Inter-American Congress of Cardiology planned for that year, the General Secretary was authorized to investigate with the Hotel Vancouver the possibility of changing the dates for the 1964 annual C.M.A. meeting.

(Continued on page 237)

NEWS & VIEWS
ON THE ECONOMICS OF MEDICINE

Prepared by the Department of Medical Economics. The Canadian Medical Association

JAN. 28, 1961, NUMBER 14

Our sources of information are private communications and published comments in medical journals and the lay press. These are usually reliable but incorrect quotation or interpretation is always possible.

"A firm stand against compulsory, government-controlled medical care insurance has been retained by the Saskatchewan College of Physicians and Surgeons.

Presenting the first submission to the Thompson Committee on medical care, the College said January 12 that medical insurance should be made available to all who want it, but only through non-government insurance plans.

The College recommended government subsidies to existing plans to reduce premiums and said measures should be taken to pay the medical expenses of Saskatchewan people who cannot meet their medical bills or join medical care plans.

The College did suggest that the medical plan in the Swift Current health region be continued---'to our knowledge . . . the only program in the world which has compulsory enrolment but no government control.'

The brief was read by College President Dr. H. D. Dalgleish of Saskatoon to the Thompson Committee, which opened hearings in the YMCA. The hearings will stretch intermittently over the next month.

Inferior Quality

'We sincerely believe,' the College said 'that a single method of insurance, compulsory for all residents and controlled by government, would result in a service of inferior quality at higher cost . . .'

The popularity of voluntary medical insurance plans was recognized.

'One cannot escape the impression that it is the success of voluntary medical services insurance that has prompted the government to extend its area of influence in this direction,' the brief said.

The College asked the government to help persons with low incomes to meet their medical services insurance costs.

'We recommend that a comprehensive medical services insurance coverage be made available to these persons on application, provided that their economic status is less than certain stated requirements. We would favor a sliding scale requirement, based upon age and number of dependents. The stated requirements would also take into consideration assets other than home or property used as a place of residence.'

Municipal officials would decide whether or not applicants from their municipalities were eligible, the College said.

Subsidize Group

'We recommend that the government subsidize almost the entire cost of providing medical services insurance to this group of persons with low salaries,' the brief said.

(over)

Few Compulsory

The brief said 56 countries in the world provide some form of medical insurance with government assistance, but very few provide a compulsory, government-controlled service.

'Within the free world, only the national health service in Great Britain provides complete coverage for all residents,' the brief said.

'Thus the introduction of a government-controlled medical services insurance program, compulsory for all Saskatchewan residents, would provide an arrangement that would differ, in varying degrees, from programs existing in many countries.'

'We must not assume that government monopoly in medical services is the arrangement found most acceptable throughout the world.'

The College favored a medical care program similar to that operating in Australia, which has a system of tax-subsidized private voluntary programs. About 75 per cent of the insurable population is covered. The patient pays the doctor and is reimbursed by the plan. The amount of reimbursement does not exceed 90 per cent of the cost.

The brief referred to an Oxford professor, John Jewkes, who has said there have been three errors in philosophy and economics behind the establishment of Britain's national health service.

Firstly, authorities greatly underestimated the cost of the health insurance, which is comprehensive and includes hospital and medical insurance.

Jewkes 'attributed this error in large part to an underestimation of the proportion of the population which would use the 'free' service,' the brief said.

The second error, according to Jewkes, was a belief that adequate medical services would ultimately make the community so healthy that medical services would hardly be needed.

Jewkes 'commented that this is the reverse of the truth and that, technically, there is no limit on the amount that a community could spend on medical care.'

'The third mistake', the College's brief continued, 'was made in supposing that the improvement in medical services would automatically increase efficiency and national income.'

Jewkes 'commented that this is not necessarily true and that, in effect, the converse is true, if the improved medical services, by lengthening life, increased the proportion of older people in the working population.'

Monopoly Employer

The College claimed that the relation between the government and the medical profession would not be one of partnership, but rather one of employer and employee.

'We would suggest that this role of 'monopoly employer' would still obtain even if the state restricted its role to one of providing payment for services, so long as the state would continue to be the sole paying agency,' the College said.

'We suggest an arrangement wherein an agreed-upon premium would be paid to existing prepayment agencies and that these agencies administer the program.'

The brief asked the Thompson Committee to consider whether or not deterrent fees have satisfied their long-term objective.

The College also suggested that the government seriously think of paying the hospitalization tax for persons with low incomes.

The College recommended that prepayment agencies be helped financially by the government in offering their medical insurance programs to individuals or groups who, because of age or physical disability, are bad risks.

Those people who are judged able to pay medical care insurance premiums from their own resources, the College said, should be encouraged to join 'the excellent programs available through the prepaid plans and the licensed insurance companies.'

'We believe these persons should have free choice to secure a coverage which meets their individual requirements,' the brief declared.

'The encouragement mentioned by the College should be health education' directed to the public, emphasizing the desirability of obtaining adequate medical services insurance coverage.'

The College suggested the government mend the health services act, so that municipalities can enter into contracts 'on a realistic basis' with medical care plans. The act currently limits municipalities to charging its residents annual premiums of more than $50 a family. The College said that existing prepayment plans charge a family from $60 to $84 a year for comprehensive coverage.

The College asked the Thompson committee to recognize the deficiencies 'which we see in existing health programs,' and suggest programs to remedy these deficiencies.

Travesty

'The treatment of the mentally ill in our institutions constitutes a travesty of a basic public responsibility . . . Hospital facilities require expansion to adequately serve current requirements.'

'Saskatchewan has a larger percentage of the population over 65 years of age than most provinces in Canada . . . The implications of our population trend have a tremendous effect upon future needs.'

The College asked substantial assistance for medical research.

'We believe this is an activity which government should encourage,' the brief said.

In the brief, the College reviewed the evolution of medical practice in Saskatchewan and set fourth 'medical milestones' in which doctors have played significant roles.

These milestones included the Anti-Tuberculosis League, the cancer commission, the establishment of the hospitalization insurance plan, and the agreement between government and doctors whereby doctors would themselves absorb part of the cost of treating social aid recipients.

(over)

In Saskatchewan, the government has made several unilateral decisions about health care without seeking the advice of doctors, or ignoring it after asking for it, the brief said.

For example, the government decided against the advice of the profession, to run the hospital insurance plan through the public health department, rather than by an independent commission.

On occasion the government has altered the social aid regulation, concerning health care received by social aid recipients, without consultation with physicians.

Discouraging

'Medical progress,' the College argued, 'has been built on the contribution of individuals. We feel the entry of government either directly or indirectly into the control of clinical medicine will discourage such individuals from entering the profession. We fear that centralization of authority and control under government, would seriously undermine this heritage.'

The College said that statements suggesting Saskatchewan's medical care plan will be similar to British and European plans have already caused alarm among provincial doctors, one third of whom are from Europe.

'This disturbance has created an unrest which is reflected in the decisions of 137 Saskatchewan doctors who left the province during the first 12 months of 1960.'

The loss figure refers to turnover of doctors, not net loss. The province had a net loss of 21 doctors in 1960.

Official registration of the College at present is 898 doctors.

'This is a direct contrast to our experience during each of the past 10 years, when the medical population of the province increased annually by an average of 31 doctors,' the brief said.

The College also added that medical students at the University of Saskatchewan are also apprehensive about the advent of government-sponsored medical care.

Students Apathetic

'The apathy of these students towards a regimented system of medical practice must accentuate a basic problem facing the profession—the decline in the number of intellectually superior students seeking entry to medicine, the brief said.

The College said it is worried that a government-sponsored medical care program would effectively retard efforts to improve the quality of medical care in Saskatchewan.

'We cannot foresee,' the brief declared, 'that the quality of medical services can possibly be enhanced under conditions which must eventually be associated with government control.

'We . . . object to the decadence in the art and science of medicine, and the monopolistic control of medical practice which we know would be the predictable outcome of a compulsory government-controlled medical services insurance.'"

REFERENCES

Regina Leader-Post, January 12, 1961.

(Continued from page 232)

Medical Care Services for Patients in Long-Term Hospitals

The Executive Committee approved the recommendation of the Committee on Economics that a joint committee of representatives from the C.M.A. and the Canadian Hospital Association be established to study the problems involved in provision of medical care services for "long-stay" patients in "chronic hospitals", the C.M.A. members to be appointed by the Chair. With regard to remuneration of physicians for services to patients in long-term hospitals, it was agreed that at this time the C.M.A. is opposed to further extension of Federal Bill 320, the Hospital Insurance and Diagnostic Services Act, to provide payment for the professional component of physicians' services.

World Medical Association

In response to a communication from Dr. Heinz Lord, Secretary-General of W.M.A., the Executive Committee voted its support of the proposal that the World Medical Association develop a permanent program designed to provide medical aid in international emergencies such as those which have presented in recent years in Hungary, Morocco and the Congo. It was also recommended that such a program should be integrated and co-ordinated with activities of other organizations in this field, such as the International Red Cross, to avoid duplication of effort and to produce the most effective medical relief program.

The Executive Committee agreed that the C.M.A. should contribute $10,000 in payment of its dues for membership in W.M.A. for 1961.

The Committee reaffirmed the appointment of Dr. Morley Young to complete his three-year term as The Association's delegate to the W.M.A. Assembly and appointed Dr. A. F. VanWart as the second C.M.A. delegate to the XVth W.M.A. Assembly at Rio de Janeiro, September 15 to 20, 1961.

Newfoundland Outport Physicians

The problems involved in settlement of the status of government-employed physicians working in cottage hospitals in the Newfoundland outports were discussed at length. To indicate The Association's support to the Newfoundland Division in its negotiations on this matter, the Executive Committee expressed the unanimous opinion that it would be unwise for the outport doctors in Newfoundland to become established civil servants and, in keeping with the recorded beliefs of the C.M.A., the Executive Committee was of the opinion that a commission type of administration of their services would be a more desirable alternative. The Committee also stated its opinion that an oath of secrecy on the part of physicians providing personal health services would be undesirable and might be disadvantageous and harmful to the quality of such services.

Submission to the Royal Commission on Government Organization

The General Secretary described certain unsatisfactory effects upon Indian and Northern Health Services that have been created by a dichotomy in responsibility for these services between the Department of National Health and Welfare and the Department of Citizenship and Immigration. The Committee approved the submission of a communication to the Royal Commission on Government Organization recommending that all government functions related to our aboriginal populations be assigned to the Department of National Health and Welfare.

The Executive Committee also:

1. Received the recommendation of the Committee on Organization that the following proposals be considered in planning future annual meetings:
   - That affiliated societies be encouraged to meet concurrently with the C.M.A. and contribute to the scientific program.
   - That scientific sessions should not overlap General Council meetings by more than one day.
   - That the C.M.A. be responsible for sessions at which endowed lectures are delivered.
   - That affiliated societies should not hold business meetings coincident with those of General Council.
   - That a Vice-Chairman be elected to relieve the Chairman of some of the duties of presiding at meetings of General Council.
2. Voted to re-confirm and leave unamended the C.M.A. Statement on Medical Services Insurance.
3. Re-affirmed approval of the establishment of a gold medal award of merit for outstanding services by members of The Association, to rank in precedence below the Starr Medal. An *ad hoc* committee of Drs. Lyon, Wigle and Lemieux was re-constituted to draft specific terms of reference and procedure for selection of recipients of such an award.
4. Approved the report of negotiations to secure an Associate Editor of C.M.A. publications and on recommendation of the Staffing Committee directed that Dr. John O. Godden of Halifax be requested to accept employment in this position on the terms outlined by the General Secretary.
5. Received the report that Dr. R. B. Raginsky of Montreal has consented to act as Chairman of a Special Committee for the Study of Hypnosis in Canada.
6. Accepted the recommendation of the Committees on Pharmacy and Public Health that the C.M.A. decline the invitation of the Canadian Public Health Association to participate in a study of problems of self-medication in Canada.
7. Authorized the General Secretary to participate in negotiations to establish a joint committee with the Canadian Pharmaceutical Manufacturers' Association to discuss mutual interests and problems, the C.M.A. representatives to such committee being Dr. Mark Nickerson, Chairman of the C.M.A. Committee on Pharmacy, Dr. E. Kirk Lyon and Dr. T. C. Routley.
8. Approved the General Secretary's action in conveying to the Minister of National Health and Welfare the concern that a rumoured discriminatory reduction in overseas living allowances for doctors in the Immigration Medical Services would constitute a threat to their morale and an obstacle to future recruitment of doctors for these duties.

Date of Next Executive Committee Meeting

The next meeting of the Executive Committee is scheduled for February 24 and 25, following the mid-winter conference of Divisional Secretaries which is to be held on February 23.

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ROYAL COLLEGE OF PHYSICIANS AND SURGEONS OF CANADA — 1960 FELLOWSHIP EXAMINATIONS

One hundred and seventy-four candidates passed the 1960 examinations of the Royal College of Physicians and Surgeons for admission to Fellowship in the College. These doctors were admitted as Fellows—a high professional honour—at the convocation which was held in conjunction with the 30th Annual Meeting of the College, held in Ottawa from January 19 to 21, 1961. The names of the 174 Fellows are listed below, according to province and city.

ALBERTA

CALGARY

Medicine—Robert Edgar Hatfield, Cameron George Hill.

EDMONTON

Medicine—Ian Hugh Holmes, David Lawrence Crawford Judge, Richard William Sherbaniuk.
Medicine (Pediatrics)—George Everett Eddy.
General Surgery—Morris Besney.
Surgery (Obstetrics and Gynecology)—Charles Alexander Douglas Ringrose.
Surgery (Orthopedic Surgery)—Glen Elmer Edwards, Alvin Hubert McKenzie.

RED DEER

General Surgery—Frans Barend Christoffel Gouws.

BRITISH COLUMBIA

NEW WESTMINSTER

General Surgery—Donald George Urlich.

TERRACE

General Surgery—Robert Edmund Musgrave Lee.

VANCOUVER

Medicine—Henry Hamon Gale, John David Ewart Price.
Medicine (Pediatrics)—Dennis Jordan Vince.
General Surgery—James Jackson Ironside.
Surgery (Urology)—John Samuel Kennedy.

MANITOBA

WINNIPEG

Medicine—David Derek Gellman, Michael John David Newman.
Medicine (Pediatrics)—Leverett Lebaron deVeber, Alvin Zipursky.
Medicine (Pathology)—Janet Elizabeth Arnott.
General Surgery—Allan Rae Downs.
Surgery (Orthopedic Surgery)—Peter Norman Porritt, Basil John Steele Grogono.

NEW BRUNSWICK

SAINT JOHN

Surgery (Urology)—Arthur Evans Chesley.

NOVA SCOTIA

HALIFAX

Surgery (Plastic Surgery)—James Frederick Ross.

ONTARIO

BREWERS' MILLS

Surgery (Obstetrics and Gynecology)—Harold Jack Kosasky.

EASTVIEW

Surgery (Orthopedic Surgery)—Denis R. DesJardins.

GALT

General Surgery—John Harris Moffat.

HAMILTON

Medicine—John Ashton Maither Henderson.

KINGSTON

Medicine—Leslie Stephen Valberg.

KITCHENER

Surgery (Obstetrics and Gynecology)—John Raymond Sehl.

LINDSAY

General Surgery—Robert James Watson.

LONDON

Medicine—Charles Hallett Lockwood.
Medicine (Pathology)—Ross George Salisbury Malone.
General Surgery—Hari Lalchand Karna, Ilgvars Henry Upmalis.

OTTAWA

Medicine—Hugh James Dempsey (presently in Birmingham, Ala.), Wallace Johnston Troup.
Surgery (Obstetrics and Gynecology)—Francis James Conklin.
Surgery (Ophthalmology)—Alexander Gardner Watson.
Surgery (Otolaryngology)—Louis-Philippe Charbonneau, Georges Guy Laframboise.
Surgery (Urology)—Patrick Gerard Murphy.

PETERBOROUGH

General Surgery—Wilbert Willoughby Belch.
Surgery (Obstetrics and Gynecology)—Alexander MacGowan Burnett.
Surgery (Ophthalmology)—Charles Edward Allen Cragg.

PORT ARTHUR

Medicine—Christopher Norrys Best, Thomas Joseph Montemuro.
Medicine (Pediatrics)—Peter Johann Koblenzer.
Medicine (Pathology)—Frank John Lone.

SARNIA

Medicine (Pathology)—Frederick Francis O'Brien.

ST. THOMAS

Surgery (Obstetrics and Gynecology)—Lillian May Beattie.

TORONTO

Medicine—Ruth Elizabeth Alison, Henry Berry, Irvin Broder, John Winniett Digby, Veronica Halmos, Caroline Emma Hetenyi, Arthur Kaminker, Morley Hart Malyon, Edward James Prokipchuk, James William Meakin, Donald Bates Montgomery.
Medicine (Anesthesia)—Brian McQuillan Marshall, Donald Irwin Matheson, Raymond Lowry Matthews, Ian Alexander Sloan.
Medicine (Neurology)—John Gilchrist Humphrey.
Medicine (Pediatrics)—Mitsumori Wesley Fujiwara, Angus Baxter MacMillan.
Medicine (Psychiatry)—Donald Blair Coate, Arthur Lambert Jones.
Medicine (Diagnostic Radiology)—Bernard Jack Shapiro.
Medicine (Therapeutic Radiology)—William John Kenneth Simpson.
General Surgery—James Coryell Fallis, John Theodore Gooding, Kenneth Weston Hobson, Irving Herschel Koven, John Alexander Macdonald, Robert Ian Mitchell, Richard Hazlewood Railton.
Surgery (Neurosurgery)—Irving Bernard Schacter.
Surgery (Obstetrics and Gynecology)—James Russell Birchard, Walter Jones Hannah, Edith Dawne Jubb, Terence Alexander Doran, Donald Charles Moore, Frank Joseph Skain, Richard Wilson.
Surgery (Ophthalmology)—Clive Bennett Mortimer.
Surgery (Orthopedic Surgery)—George Gordon Dale, Michael Andrew Simurda.
Surgery (Plastic Surgery)—Erwin Mitchell Tanz, Hugh Gordon Thomson.
Surgery (Urology)—Vincent Colapinto, Francis Mary Michael O'Kelly.

WINDSOR

Medicine—Cameron Dewar Anderson.
Surgery (Obstetrics and Gynecology)—Andrew Ammud Freier.

QUEBEC

ALMA (LAC ST-JEAN)

General Surgery—Gérard Magella Couture.

CHICOUTIMI

Medicine—Léo Gosselin.
Medicine (Therapeutic Radiology)—Léopold Genest.

DRUMMONDVILLE

General Surgery—Joseph Bernard Jean Guy Paillé.

HULL

Medicine (Pathology)—Jean Louis François Sirois.

LÉVIS

Surgery (Obstetrics and Gynecology)—Jean Turmel.

MONTREAL

Medicine—Richard Edgar Donevan, Joseph Fishman, Richard Roblin Gillies, Duncan Alexander Gordon, Carl Arthur Goresky, Yvon Goulet, Harvey Zachary Hollinger, Michel Lacombe, Arthur Leznoff, William Edward Halton Mason, Pierre Nadeau, André Proulx, Jean Guy Queeneville, Jacobo Isaac Teitelbaum, Florent Thibert, Isadore Wayne Weintrub.
Medicine (Anesthesia)—Frederic Robert Holland Wrigley.
Medicine (Dermatology)—Thomas Joseph Sullivan.
Medicine (Neurology)—André Barbeau.
Medicine (Pediatrics)—F. Luc Chicoine, Gloria Jeliu.
Medicine (Diagnostic Radiology)—Fleming McConnell, George Bernard Skinner.
General Surgery—Claude Bergeron, Jean Gauthier Desjardins, Frederic Graham Inglis, Jean-Louis Leclerc, Geoffrey William Lehman, Latiff Georges Sarkiss, Jean-Guy Vallée.
Surgery (Obstetrics and Gynecology)—Roland Peter Beck.
Surgery (Ophthalmology)—Jean Dumas.
Surgery (Orthopedic Surgery)—Frederick Alan Hamilton Greenwood.
Surgery (Otolaryngology)—Yoginder Nath Mehra.

PONT VIAU

Surgery (Orthopedic Surgery)—Raymond Lemaire.

QUEBEC

Medicine—Raymond Laflamme, Joseph Rodolphe Guy Saucier.
Medicine (Pathology)—Léonard Bernier, Paul Maurice Gagnon.
General Surgery—Raymond-Marie Dion, Camille Gosselin, Paul-Emile Patry, J. Maurice Vignault.
Surgery (Orthopedic Surgery)—Jean-Jacques Ferland.

RIMOUSKI

Surgery (Obstetrics and Gynecology)—André Hubert Bernier.

SHAWINIGAN FALLS

General Surgery—René-Georges Tremblay.

SHERBROOKE

Medicine (Pediatrics)—Bernard Therien.
General Surgery—Louis-Philippe Lejeune.

VILLE ST-LAURENT

Medicine (Pediatrics)—Peter Benjamin.
Medicine (Pathology)—George Rona.
Surgery (Otolaryngology)—William Henry Novick.

SASKATCHEWAN

SASKATOON

Medicine—Marcel Alter Baltzan, Robert Neil Beck, Arthur Elwood Somerville.
General Surgery—Alexander Pollock.

UNITED STATES OF AMERICA

WASHINGTON, D.C.

Medicine—Anandeswar Barthakur.

MCHENRY, ILLINOIS

Medicine—Benjamin Jacob Massouda.

DES MOINES, IOWA

Surgery (Urology)—Dieter Kirchheim.

BOSTON, MASSACHUSETTS—

Medicine—Frank Alexander Herbert, William Allan Mahon.

HEMPSTEAD, NEW YORK

Surgery (Ophthalmology)—Martin Stephen Kazdan.

CLEVELAND, OHIO

Medicine—Edmond Joseph Goold.

PHILADELPHIA, PA.

Surgery (Ophthalmology)—Hedwige Elfriede Chodos, Joel Boaz Chodos.

SEATTLE, WASHINGTON

Medicine—Walter Charlton MacDonald.

JAMAICA, WEST INDIES

General Surgery—Vivian Earl Hamilton Brooks.

INDIA

Surgery (Urology)—Balchand D. Bapna.

PUBLIC HEALTH

PARALYTIC POLIOMYELITIS IN CANADA*
50TH WEEK—ENDING DECEMBER 17, 1960

	Reported cases									*Deaths*		
	This week			*Last week*			*To this date*			*To this date*		
	1960	*1959*	*1958*	*1960*	*1959*	*1958*	*1960*	*1959*	*1958*	*1960*	*1959*	*1958*
Canada	14	7	1	11	6	3	820	1852	245	72	177	25
Newfoundland	—	—	—	1	1	—	49	139	4	4	12	—
Prince Edward Island	—	—	—	—	—	—	1	7	—	—	1	—
Nova Scotia	—	—	—	—	—	—	9	8	—	1	—	—
New Brunswick	—	—	—	—	—	—	50	61	4	2	6	1
Quebec	7	5	—	7	3	1	248	1151	76	33	104	5
Ontario	—	—	—	—	—	—	39	198	20	2	21	5
Manitoba	—	—	1	—	—	—	12	26	106	1	2	10
Saskatchewan	3	—	—	—	—	—	53	41	1	8	3	—
Alberta	4	—	—	2	1	—	193	80	22	9	12	1
British Columbia	—	2	—	1	1	2	165	130	11	12	12	3
Yukon	—	—	—	—	—	—	—	—	—	—	—	—
Northwest Territories	—	—	—	—	—	—	1	11	1	—	4	—

*Weekly returns based on telegraphic reports by provinces to the Dominion Bureau of Statistics.

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World Medical Association

The World Medical Association recently announced the appointment of a new Secretary General to replace Dr. Louis H. Bauer, who has retired from this position. The new appointee is Dr. Heinz Lord, recently of Barnesville, Ohio, and now resident in Sea Cliff, New York.

Dr. Lord has an interesting international background. Born a Peruvian citizen, he was brought up and educated in Hamburg, Germany, and Zurich, Switzerland. During the Second World War, being anti-Nazi, he was placed in a concentration camp. Near the end of the war he was on board a steamship, containing 800 internees, which was bombed in the Baltic Sea. Dr. Lord was one of 28 survivors.

Joseph Merante, Jr., N.Y.

Dr. Louis H. Bauer (right), retiring Secretary General of the World Medical Association, congratulating Dr. Heinz Lord on assumption of that office.

Returning to Hamburg at the end of the war, he was active in organizing the Marburger Bund.

In 1954 he migrated to the United States. After three years of further postgraduate training, he entered surgical practice in Barnesville, Ohio. He is a member of the American Medical Association, a Fellow of the International College of Surgeons and a member of the German Urological Society.

Dr. Bauer became Secretary General in April 1948, shortly after the Association was organized. During his tenure the World Medical Association increased its membership from 21 to 56 countries.

Dr. Bauer, a resident of Rockville Center, New York, is a Past President of the American Medical Association and a former Chairman of the Board of Directors of United Medical Service, Inc. (Greater New York's Blue Shield Plan).

While Secretary General, he made 26 trips to Europe, two around the world, two to Australia, two to the Caribbean area and one to South America, visiting in all over 40 countries, in an effort to unite the medical profession of the world in bringing about higher standards in medical care, medical education and public health.

On his retirement Dr. Bauer became a consultant to the World Medical Association.

BOOK REVIEWS

SYNOPSIS OF PATHOLOGY. W. A. D. Anderson. 414 pp. Illust. 4th ed., The C. V. Mosby Company, St. Louis, Mo., 1960. $9.25.

In its new edition this previously well-accepted book remains a concise but comprehensive presentation of those aspects of pathology that are of particular value to the student, undergraduate or postgraduate, wishing a quick review in which the broad outlines and patterns of disease are unobscured by a maze of detail.

The contents are arranged as in most standard textbooks of pathology, into 11 chapters on general pathology followed by 14 chapters on systemic pathology. In the present edition most of the chapters have been subjected to some revision. Reference, although necessarily brief, is made to many aspects of the changing significance and concepts of our knowledge of disease. Cognizance is taken of the changing significance and prominence of some viral infections, and of new information regarding a number of conditions such as pulmonary alveolar proteinosis, pneumocystis pneumonitis, kwashiorkor, epidemic hemorrhagic fever, aldosteronism, carcinoid syndrome, toxoplasmosis, nephropathy of potassium deficiency, uremic pneumonitis, atypical hyperplasias of the lung, the pathogenesis of Hashimoto's disease, the relationship of giant cell pneumonia and measles and many other subjects of current interest or recent advent.

The book contains 414 photographs, most of which are of good quality and well selected to illustrate particular points. A number of tables are used to compare and contrast related diseases or processes in synoptic form.

Finally, it is to be noted that this synopsis is in reality a résumé of one of the finest detailed textbooks in pathology, edited by Dr. Anderson in association with 31 outstanding collaborators. This synopsis reflects the soundness of the larger volume, and for its special purpose this book can be highly recommended.

INTERNAL MEDICINE: A Physiologic and Clinical Approach to Disease. R. P. McCombs, Professor of Graduate Medicine, Tufts University School of Medicine. 750 pp. Illust. 2nd ed. Year Book Publishers, Inc., Chicago, 1960. $10.50.

The author has provided his answer to a question which is vexing many internists, "What is an internist and what constitutes internal medicine?" The answer does not come in a short sentence which might inform the public or satisfy insurance companies and governmental agencies. The answer is the book itself. Not all would agree, however; in particular, one is surprised to find neurology omitted and even more surprised that the subject of internal medicine could be covered without a separate section on psychiatric disturbances which commonly present with somatic symptoms, e.g. anxiety states and depression. Few internists would deny that much of their task (and often the most difficult part) consists of recognizing and treating psychological disturbances.

The book is compact, economical of words and, what is rather rare but highly commendable, the work of a single author. There is always room for the good medical writer who organizes and clearly presents those principles, theories and observations which he uses in

ay-to-day practice. This book would be useful to student or recent graduate considering internal edicine as a career and wishing to find out what an ternist reasonably should be expected to know. The b-specialist or general practitioner, or the specialist another field altogether, would benefit by reading s up-to-date conservative account of internal medie. Rather than a gigantic reference book, of which ere are a number, it is a work of which there are ll too few, presenting the essentials of the subject. Most general internists will find the contents of the ook quite familiar, and for help with difficult cases vill turn to other more detailed works.

TLAS DER GYNAEKOLOGISCHEN ANATOMIE. Heinrich Martinus und Käthe Droysen. 118 pp. Illust. Georg Thieme Verlag, Stuttgart, W. Germany; Intercontinental Medical Book Corporation, New York, 1960. $11.65.

This new atlas of gynecological anatomy has grown out of the work previously published by the same authors in their textbooks on obstetrics and gynecology. The atlas is divided in two chapters: one is a text of 40 pages, and the other contains 78 pages of illustrations.

The text may be considered as a summarized discussion of the anatomy of the female pelvis and pelvic organs. A short description of the hypophysis and the peripheral nervous system in the female pelvis is also given. This text is hardly adequate for either the specialist in this field or for the anatomist. However, it is useful for the general practitioner who now and then may be confronted with gynecological problems, diagnostic as well as therapeutic. The illustrations are fair from the point of view of art and do not give more detail than is necessary for a general review. The pictures of the ovary and particularly those of the developing ovum and blastocyst are a favourable exception. The explanation of the figures in three languages, German, English and Spanish, is interesting and may be useful for the ultimate distribution of the atlas.

The price of the book is rather high.

ELEKTROLYT-KOMPENDIUM (Electrolyte Compendium). W. Fleischer and E. Fröhlich. 345 pp. Illust. Benno Schwabe & Co. Verlag, Basle and Stuttgart; Intercontinental Medical Book Corporation, New York, 1960. $9.50.

This well-organized book, written in German, compiles what is known to date about water and electrolytes in the human body. The first three chapters are devoted to basic facts on physics, chemistry and physiology, so that the deviations from normal which follow can be more readily understood.

Part II begins with a chapter on diagnosis, which leads into an excellent discussion of replacement therapy in all of its theoretical and practical aspects. Subsequent paragraphs deal with water and electrolyte imbalance in relation to endocrine glands, vitamins, diabetes mellitus, cardiac disease and renal disorders. Finally, the special problems of surgical patients and children are summarized.

The illustrations and tables are clear and instructive, and a good index and an extensive bibliography add to the value of this book. Anyone who wants to acquire a thorough knowledge of the subject will appreciate this volume, and it will be found equally useful for quick reference in practical problems.

(Continued on page 244)

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(Continued from page 241)

AN APPROACH TO OCCUPATIONAL THERAPY. Mary S. Jones. 245 pp. Illust. Butterworth & Co. (Canada) Ltd., Toronto, 1960. $8.50.

In this book the author reviews the activities of the occupational therapy department of the Resident Rehabilitation Centre in Slough over eight years from its inception in 1947 to 1955. The centre is essentially for ambulatory, short-term cases (maximum ten weeks) with expectation of "a quick return to a normal life in a professional or industrial capacity". Mrs. Jones reviews the various disabilities seen according to whether they involve the spine, upper limbs or lower limbs and also includes chapters on abdominal disease, disabilities of lungs and thorax and so forth.

The occupational therapy approach to various disabling conditions is discussed and a chapter is devoted to workshop techniques in which ingenious adaptation of equipment and machines, aimed at restoring function to the disabled patient, is described.

Unfortunately, the overall therapeutic results of treatment are not well documented although many individual success stories are cited. This lack of an objective index of evaluation of the application of occupational therapy skills tends to detract from the scientific value of this survey. At times the book is repetitive and the program not clearly delineated. However, certain sections give a clear picture of the techniques embodied in a progressive activity program; e.g. in the sections dealing with post-meniscectomy and post-arthroplasty patients.

The author has sprinkled the book with anecdotes which reveal the understanding, personal philosophy and enthusiasm with which she has approached the interesting problems met in the Resident Rehabilitation Centre.

The book would appear to be pointed particularly to the student of occupational therapy. Nonetheless, it will give insight to the interested physician into the functional approach to treatment as embodied in the profession of occupational therapy.

INTERNATIONALES BIGUANID-SYMPOSIUM. 12 und 13 Mai 1960 in Aachen. Prof. F. Bertram. 164 pp. Illust. Georg Thieme Verlag, Stuttgart, W. Germany; Intercontinental Medical Book Corporation, New York, 1960. $4.00.

Symposia are a popular and useful method for the dissemination of knowledge accumulated in various centres regarding a drug or a group of drugs, and such a symposium is presented by the editors of this volume devoted to the subject of the biguanides. The chemistry and pharmacology of these agents were presented by renowned investigators from various centres in Germany. Discussion followed the more formal presentations in this and in the other portions of the symposium. Regarding the toxic and side effects of biguanides, there was, by and large, general agreement that these increase with higher dosage and have been considerably reduced in frequency since dosages of 150 mg. per day or less have been used. Of some interest is the suggestion advanced by several speakers that there is a parallelism between the incidence of side effects, especially those affecting the gastrointestinal tract, and the effectiveness of the biguanides in reducing blood sugar levels. Several cases of Ménière's syndrome were reported, indicating that side effects may involve the central nervous system or may possibly be due to an adrenaline-like action of the drug.

The experiences of American workers were presented by H. S. Sadow of New York, and by Krall, Bradley and Priscilla White, of the Joslin Clinic. There followed extensive reporting of personal experiences with both DBI and with DBV (W37 or n-butylbiguanide) by a number of leading German diabetologists, and a most interesting practical approach to treatment wth biguanides on an outpatient basis, by Dr. Curchod of Lausanne, Switzerland. The mode of action of biguanides is still far from elucidated and the tantalizing question still remains why they have hardly any hypoglycemic effect on normal subjects.

There was almost unanimous agreement regarding the indications for the use of DBI. Its main usefulness is for the juvenile, unstable diabetic, for the aged diabetic whose beta cells have become atrophied because of prolonged insulin administration and for the insulin-deficient asthenic elderly person. It will have occasional value for patients with insulin resistance and for those with sensitivity to insulin. The American expression, "smoothing" effect of biguanide in labile diabetes, has been readily accepted by German workers. They also agree that the "go slow" method of initial administration helps to prevent intolerance to biguanides.

Both Bertram and Steigerwald stressed the point that even at this stage of incomplete knowledge concerning the long-range effects of biguanides, they would not like to be without these drugs in treating diabetic patients.

BEHANDLUNG INNERER KRANKHEITEN. Richlinien und Ratschläge für Studierende und Aerzte. Prof. Ferdinand Hoff. 872 pp. 9th ed. Georg Thieme Verlag, Stuttgart, W. Germany; Intercontinental Medical Book Corporation, New York, 1960. $14.15.

This is the ninth edition of a time-tested text on therapy in internal medicine, written in German. The nearly 900-page volume not only fulfils such essential requirements as quick and complete indexing, but is well up to date in critical evaluation of modern therapy.

The tremendous number of new remedies makes it impossible for the practising physician to be more than superficially acquainted with all of them. The authors have made their selection according to their own obviously wide clinical experience. By necessity the book is to some degree authoritative in this selection. The authors emphasize the importance of the physician's complete familiarity with the drugs he prescribes.

The various chapters are written by experienced clinicians, working in fields they describe. The book reaches far beyond mere methods. It provides a form of often philosophical, humanistic guidance, accentuating the all too often forgotten fact that more than chemical compounds are needed to heal. Several introductory chapters are devoted to therapeutic planning, according to the individual patient's physical and economic status.

Most interesting reading is provided in chapters which concern themselves with treatment of iatrogenic and drug-induced diseases and with vegetative system disorders. Of much value is a specially appended outline of treatment in cases of poisoning.

This book will be welcomed for guidance and reference by the medical student, general practitioner or specialist.

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PROCEEDINGS OF THE ELEVENTH ANNUAL CONFERENCE ON THE NEPHROTIC SYNDROME. Edited by Jack Metcoff. 324 pp. Illust. National Kidney Disease Foundation, New York, 1960.

The eleventh annual conference on the nephrotic syndrome was held at the Johns Hopkins Hospital in October 1959. This conference was more comprehensive in its scope than the title implies. The papers presented are grouped under five main headings: (1) The role of counter current mechanisms in urine concentration. (2) Renal reabsorption of sodium. (3) Infection and renal disease. (4) Research in progress. (5) Therapy of the nephrotic syndrome.

In Section I there are papers by such eminent authorities as Ullrich, Schmidt-Neilsen and Gottschalk. In Section II there is an excellent paper by Leaf on the effects of neurohypophyseal hormones on the living membrane. Section III deals with a wide variety of subjects including intrarenal infections, immunogenic renal disease and extrarenal infection, serum complement in experimental nephritis, the pathogenesis of pyelonephritis and studies of renal function in the chronically diseased kidney. Section IV includes three papers on the electron microscopy of the kidney along with many excellent electron micrographs.

Each topic is dealt with by the experts in that particular field and the group discussions of the various reports are recorded. This volume is recommended to everyone interested in kidney function and disease, be he basic scientist or clinician.

DAS MENSCHLICHE KNOCHENMARK (The Human Bone Marrow). Karl Rohr, Zürich, Switzerland. 593 pp. Illust. 3rd ed. completely revised. Georg Thieme Verlag, Stuttgart, W. Germany; Intercontinental Medical Book Corporation, New York, 1960. $23.35.

This monograph on the human bone-marrow is a Swiss work written in German. It is in its third edition and has been extensively revised and brought up-to-date.

A short outline of the history of bone-marrow examination leads into a description of past and present methods of securing specimens and their preparation for study. The next chapters give a detailed account of the anatomy and physiology of the hematopoietic system in the broadest sense of the word. This, of course, brings up a number of controversial points, as some phases of the hematopoietic process are not yet completely understood. The different opinions of the leading hematologists are discussed and evaluated on the basis of facts recently brought to light by new methods of research. This is followed by a systematic and exhaustive presentation of the primary and secondary pathological conditions of the blood and the bone marrow, which is based on the author's own observations as well as a thorough study of the world literature. The material is handled from the pathological and clinical point of view; in fact it often reads like a text on medical disorders as reflected by changes in the hematopoietic system.

"The Human Bone Marrow" is a very modest title, indeed, and both the pathologist and the internist will find a wealth of information in this volume. The good print and the beautiful illustrations add to the value of the book, as does the excellent bibliography which comprises 50 pages of small print.

Books Received

Books are acknowledged as received, but in some cases reviews will also be made in later issues.

Situation et Valeur de la Psychologie en Médecine. G. Berger, W. Bovan, P. Mayrac et L. de Pina. Collection "Psychologie Médicale" sous la direction de A. Lamache et P. H. Davost. 107 pp. L'Expansion Scientifique Française, Paris, 1960. 5 NF. (approx. $1.00).

Précis de Proctologie Infantile. A. Bensaude et P. L. Chigot avec la collaboration de A. Roberti. 120 pp. L'Expansion Scientifique Française, Paris, 1960. 11.50 NF. (approx. $2.25).

L'Approche Psycho-Sociale en Médecine. G. Huyer, A. Missenard, P. Pichot et R. Quero. Collection "Psychologie Médicale" sous la direction de A. Lamache et P. H. Davost. 59 pp. L'Expansion Scientifique Française, Paris, 1960. 5 NF. (approx. $1.00).

Controlled Clinical Trials. Papers delivered at the Conference Convened by The Council for International Organizations of Medical Sciences. Organized under the direction of Professor A. Bradford Hill. 177 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $6.00.

Dietary Proteins in Health and Disease. James B. Allison and William H. Fitzpatrick. 86 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $5.00.

The Chemistry of Thyroid Disease. Rosalind Pitt-Rivers and Jamshed R. Tata. 83 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $5.00.

Proceedings of the Eleventh Annual Conference on the Nephrotic Syndrome. Edited by Jack Metcoff. 324 pp. Illust. National Kidney Disease Foundation, New York, 1960. $5.25.

Cardiac Disease in Pregnancy. Curtis Lester Mendelson. 385 pp. Illust. F. A. Davis Company, Philadelphia; The Ryerson Press, Toronto, 1960. $14.75.

A Minority. A report on the life of the male homosexual in Great Britain. Gordon Westwood. 216 pp. Longmans, Green & Company, Toronto, 1960. 30s. net.

The Rudolph Virchow Medical Society in the City of New York 1860-1960. Edited by Joseph Berberich, Henry Lax and Rudolph Stern. 523 pp. Illust. S. Karger, Basel; Albert J. Phiebig, White Plains, N.Y., 1960. $16.00.

Hypnosis in Skin and Allergic Diseases. Michael J. Scott. 161 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $7.25.

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GENERAL SURGEON, 31, Canadian, American trained. Fully qualified. Wishes to locate an associate practice, or group practice. Available July 1 1961. Reply to Box 215, CMA Journal, 150 St. George Street, Toronto 5, Ontario.

BRITISH DOCTOR, married, 38, postgraduate with wide experience in Canada and Britain seeks assistantship with view or junior partnership in Toronto, within vicinity of Forest Hill area. Able to contribute to practice or make working arrangement. Reply to Box 255, CMA Journal, 150 St. George Street, Toronto 5, Ontario.

30-YEAR-OLD Canadian graduate who has completed 1 year junior rotating internship at the Montreal General Hospital is interested in obtaining a locum or assistantship, preferably in Ontario or Quebec, from August 1, 1961 to December 31, 1961. Reply to Box 256, CMA Journal, 150 St. George Street, Toronto 5, Ontario.

ALBERTA GRADUATE, married with family, desires permanent association with group offering opportunities for future postgraduate study. 2 years internship, 4 years successful general practice with considerable experience in anesthesia. Reply to Box 258, CMA Journal, 150 St. George Street, Toronto 5, Ontario.

MEDICAL NEWS *in Brief*

(Continued from page 230)

GASTRECTOMY FOR ULCER BESET BY SERIOUS PROBLEMS

Subtotal gastrectomy for duodenal ulcer was characterized as a "highly unsatisfactory procedure" in a review of 300 gastrectomies reported to the 25th annual scientific session of the American College of Gastroenterology in Philadelphia. Ricketts of New Jersey and Straub of Philadelphia said that the procedure is "one beset by many serious problems from the initial selection of the patient for surgery throughout the remainder of his life". The conclusions were drawn from a study of unselected patients at the gastrointestinal clinic of the Veterans Bureau Regional Office in Philadelphia. The two criteria were that the patients had undergone subtotal gastric resection for duodenal ulcer and that the postoperative period at the time of study be one year or more.

Evaluating overall results, they found that the condition of 103 of the patients (34.3%) could be considered good; of 133 (44.3%), fair; and of 64 (21.4%), poor. But where mental or emotional problems were involved – as they were in 108 patients – the results were good in only 21 patients (19.5%); fair in 52 (48.1%); and poor in 35 (32.4%). Moreover, a review of the socioeconomic status of the patients indicated that only 50.3% had returned to their original work, while 33.7% had to take lighter jobs, and 15% had not returned to work at all – in postoperative periods ranging from one to 11 years. The remaining 1% were studying under the G.I. Bill of Rights.

In another report, Dr. Everett D. Kiefer, chairman of the Department of Gastroenterology, Lahey Clinic, Boston, said that patients with postgastrectomy syndrome require individualized treatment; hospitalization was recommended when symptoms are severe. Regulation of diet and eating habits was singled out as the most important feature of treatment. Small quantities of food taken at frequent intervals provide a more adequate daily ration for such patients. Psychotherapy also plays a role in treatment, and patients should receive reassurance, an explanation of the cause of their symptoms, and directions for stopping aerophagia. – *Medical Tribune,* November 14, 1960.

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KERATOCONJUNCTIVITIS SICCA AND SJOGREN'S SYNDROME

Interest in keratoconjunctivitis sicca, an inflammation of the cornea and conjunctiva associated with dryness, was primarily confined to ophthalmologists until 1933 when Sjögren described the combination of stomorhinopharyngolaryngitis sicca, salivary gland enlargement and arthritis in patients with keratoconjunctivitis sicca, a combination since designated as Sjögren's syndrome.

The records of 248 patients on whom this diagnosis had been established at the Mayo Clinic from 1950 to 1956 were studied, and a questionnaire follow-up

(Continued on page 21)

Effective penicillin therapy with just one tablet every 12 hours

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Bottles of 10 tablets.

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*Pat. 1959

CAUTION: In rare instances, the injection of penicillin, and more rarely still its oral administration, may cause acute anaphylaxis. The reaction appears to occur more frequently in patients with bronchial asthma and other allergies, or in those who have previously demonstrated sensitivity to penicillin.

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MEDICAL NEWS *in brief*

(Continued from page 19)

was answered by 138 patients of this group. In addition, a follow-up questionnaire was submitted to a series of 121 other patients with this syndrome who had been subjected to a previous study. Of these, 59 replied to the questionnaire. The present study was undertaken to investigate the relationship of other clinical conditions to keratoconjunctivitis sicca and Sjögren's syndrome and to determine the occurrence of various protein and hematological abnormalities in both conditions. The incidence of the different individual manifestations of the syndrome among patients with keratoconjunctivitis sicca was explored and the course and prognosis of both of these disorders was studied (C. A. Stoltze, D. G. Hanlon, G. L. Pease and J. W. Henderson, *A.M.A. Arch. Int. Med.*, 106: 513, 1960). The following observations were made:

The proportion of men to women was higher than usually reported. In this series, 18% of the patients were men. The low incidence of males in other reports may be related to the fact that Sjögren's syndrome tends to be milder and less easily recognized in the male.

The frequency of arthritis was less than is commonly reported. In this study the term "arthritis" was confined to joint disease of rheumatoid type accompanied by evidence of systemic disease. Keratoconjunctivitis sicca was said to be the most common ocular disorder of patients with rheumatoid arthritis but the nature of the relationship between the joint and ocular manifestations is not clear. The exclusion of other types of joint disease in this study possibly accounts for its lower incidence of "arthritis" (21%) compared with an incidence of 45% reported in a total of 589 cases from several of the larger series published previously.

Conditions most frequently associated with keratoconjunctivitis sicca and Sjögren's syndrome were related to dryness of mucous membranes of the oropharynx and of the respiratory and gastrointestinal tracts.

Associated conditions not related to dryness of mucous membranes included thyroid diseases (toxic nodular, adenomatous and exophthalmic goitre, thyroiditis, myxedema and carcinoma), "collagen diseases" other than rheumatoid arthritis (7%), and the hyperglobulinemic purpura of Waldenström (observed in 9 cases of Sjögren's syndrome).

A variety of protein and hematological abnormalities was encountered in patients with Sjögren's syndrome.

There was no evidence to suggest a direct relationship of keratoconjunctivitis sicca to ovarian or adrenocortical dysfunction.

The follow-up study indicated that although the prognosis for cure is poor at present, both keratoconjunctivitis sicca and Sjögren's syndrome, while characterized by remissions and exacerbations, do not apparently tend to progress over a number of years in a high percentage of cases, and have a good prognosis.

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INTERNATIONAL CONFERENCE ON LIVING CONDITIONS, HEALTH AND ECONOMIC DEVELOPMENT

The Third International Conference of the International Medical Association for the Study of Living Conditions and Health will be held from September 29 to October 1, 1961, in St. Vincent, Aosta Valley (near Turin), Italy, under the title "Living Conditions, Health and Economic Development." The preliminary program is as follows:

I. *Public Health as a Factor in Economic Development*

1. Rural Endemics and Agricultural Productivity.

2. The Organization of Rural Medical Services and Economic Development.

II. *The Influence of Economic Development on Health*

1. Development of Agrarian Economy and the Fight Against Rural Endemics.

2. Industrial Development and its Eventual Hazards for Public Health.

III. *Conclusions: Health Problems and Planning of Economic Development.*

Further information about the Conference can be obtained from the General Secretary, International Medical Association for the Study of Living Conditions and Health, Burggasse 71/I/6, Vienna 7, Austria.

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